Pathological Anatomy,

PATHOLOGY AND PHYSICAL DIAGNOSIS.

A Series of Clinical Reports

COMPRISING THE

PRINCIPAL DISEASES OF THE HUMAN BODY.

Systematically arranged in One Hundred Full-page Illustrations and One Hundred Pages Text.

BY J. A. JEANÇON, M. D.

Author of "Atlas of Human Anatomy."

CINCINNATI, O.

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INTRODUCTORY.

Being the first to undertake the publication, in this country, of a work like the present, we have reason to anticipate a favorable reception.

The author, in this series of clinical histories, presents a resume, concise, yet sufficiently comprehensive of the data of pathological anatomy of the principal diseases of the human body, as evolved from the great schools on the European Continent, and England, since the early part of this century, when modern medicine was established upon a solid basis, by direct clinical observations and postlethal examinations of the morbidly changed structure, up to the present time. Selecting many cases from standard works, and current medical literature, he made them the central portion of his text, and very carefully delineated the pathological features of the class of which each narrative forms a clinical type.

The names of a great number of authors consulted by him and referred to in this work, are given on pages five and six as well as in the body of the text. The latest and the firmest established medical facts are incorporated in the pathological and diagnostic parts of the work. He endeavored chiefly to place before the profession, portions of the vast collections of the labors of the great teachers, existing in the shape of highly instructive illustrated works, still only in few great medical public libraries, and on the shelves of some few physicians peculiarly favored by good fortune, but inaccessible, by reason of their high price and the scarcity of published copies, to the bulk of the profession.

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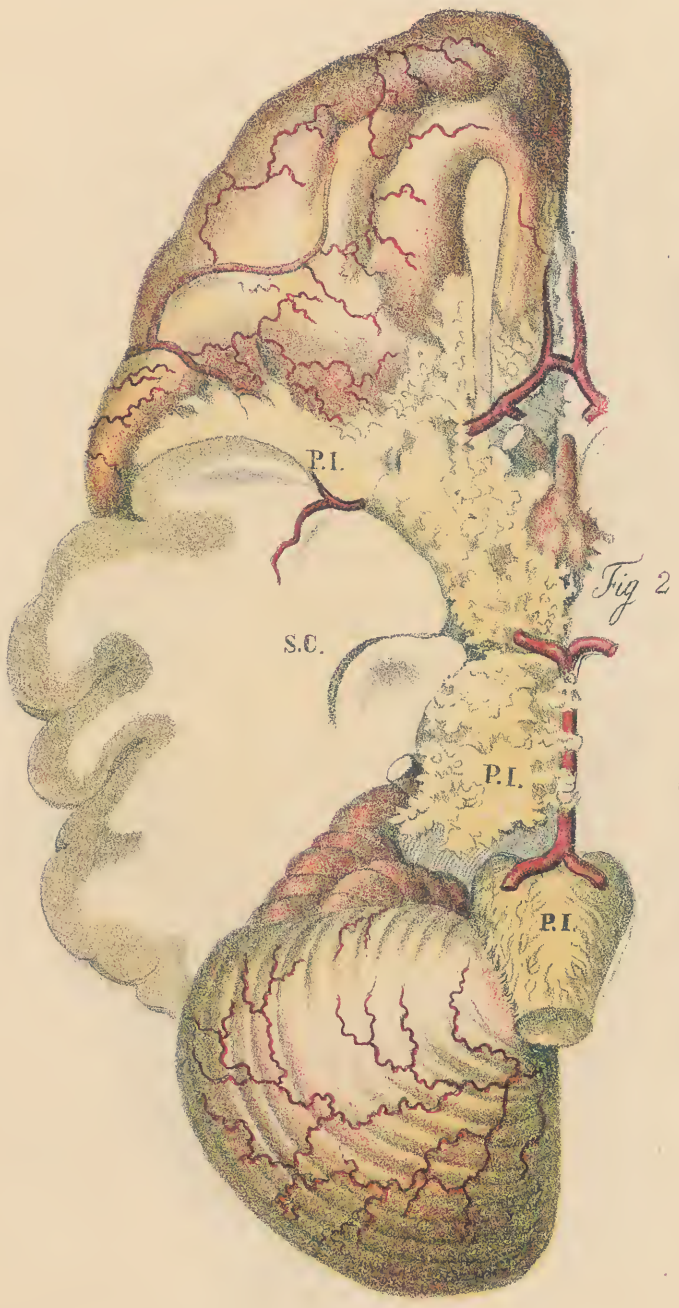
CEREBRAL ARACHNITIS.

Sec. I. *Tab. 1.*



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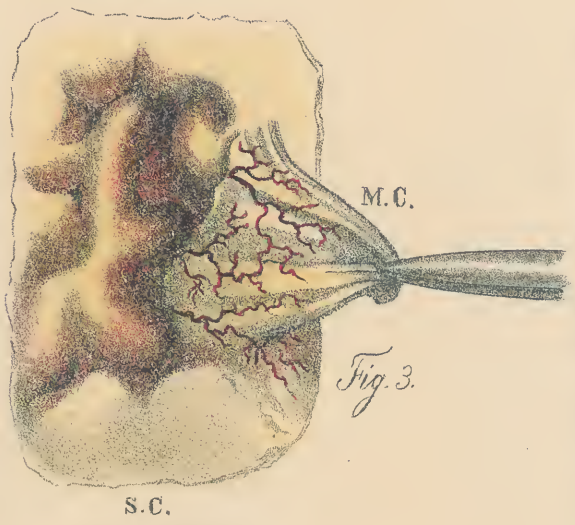

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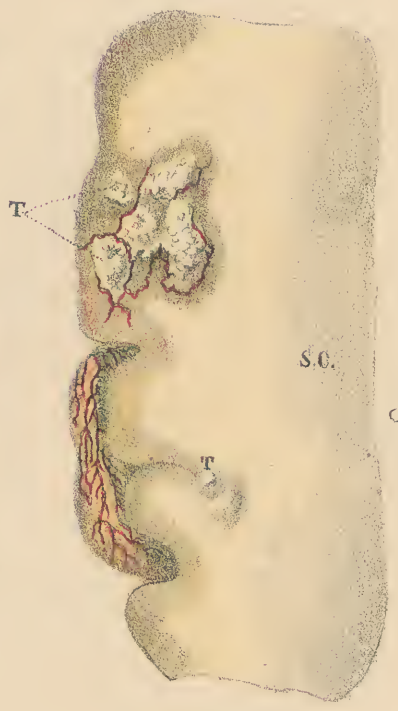

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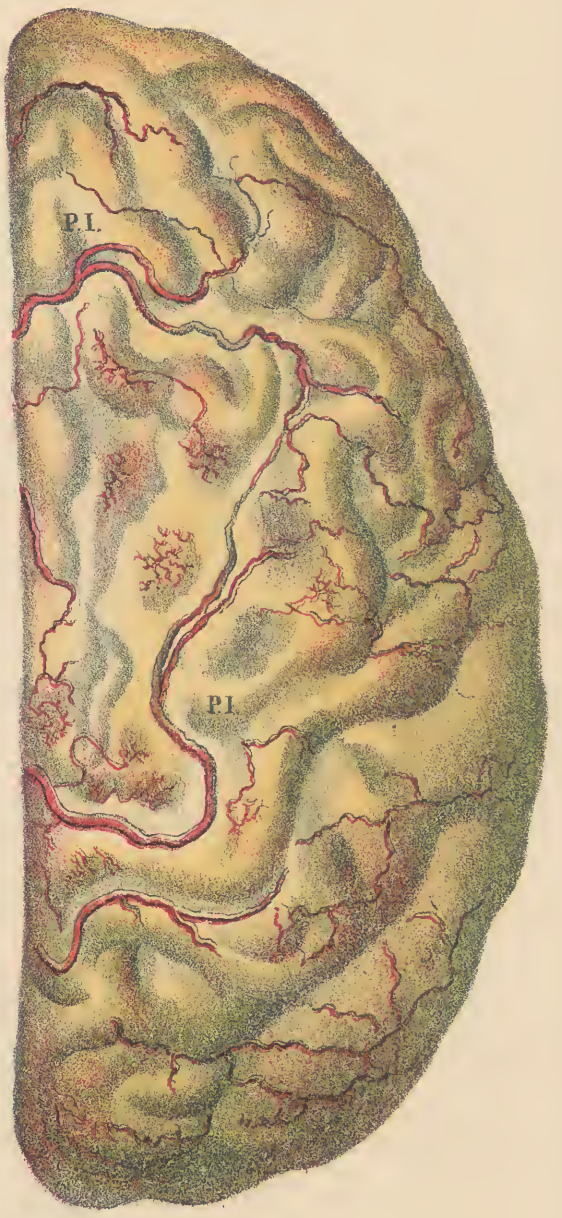

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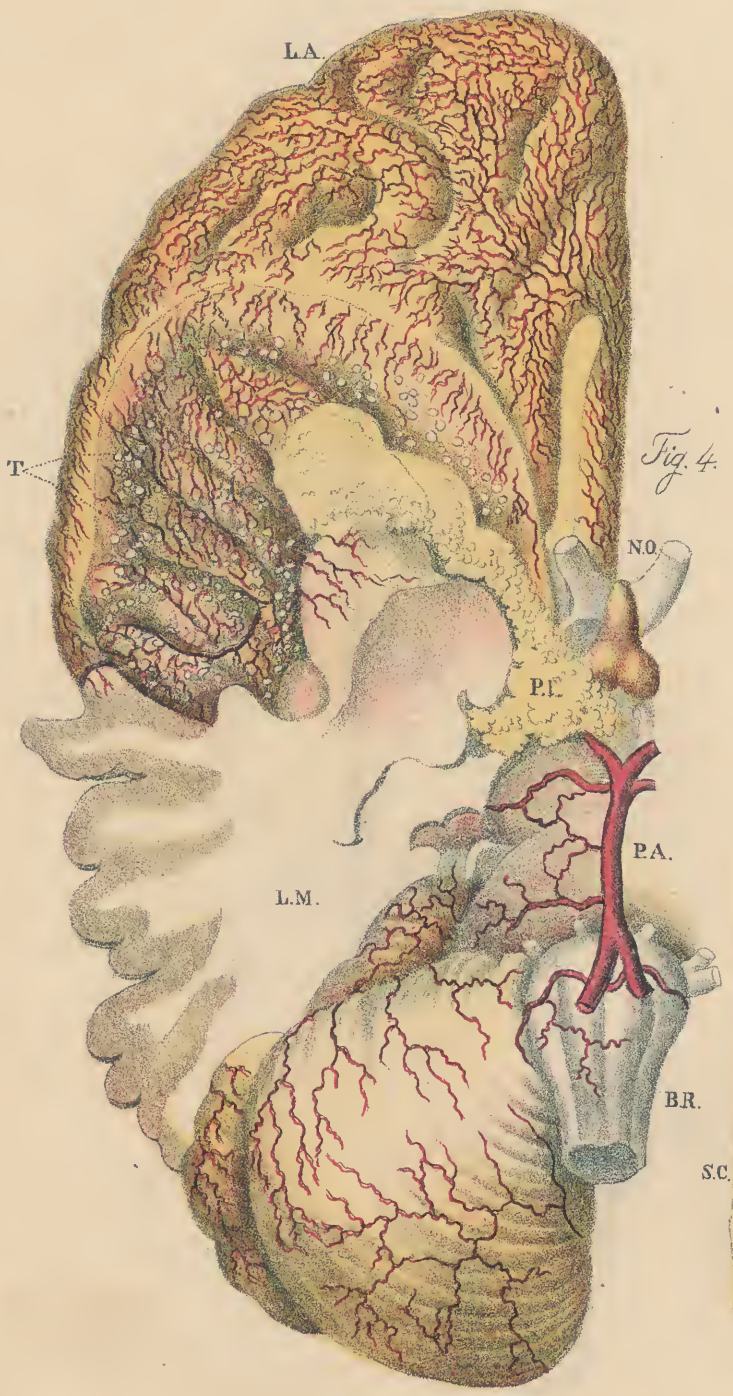

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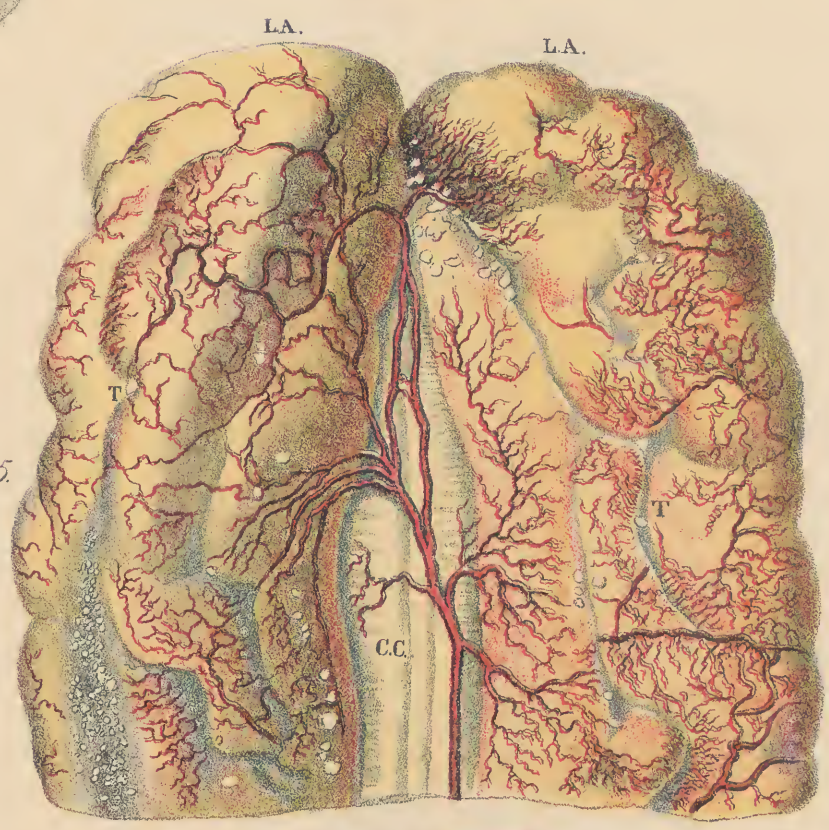

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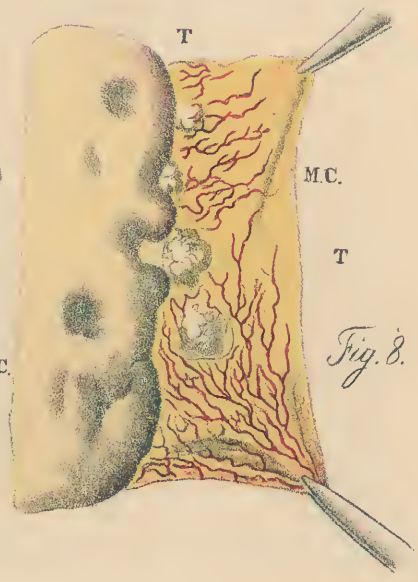

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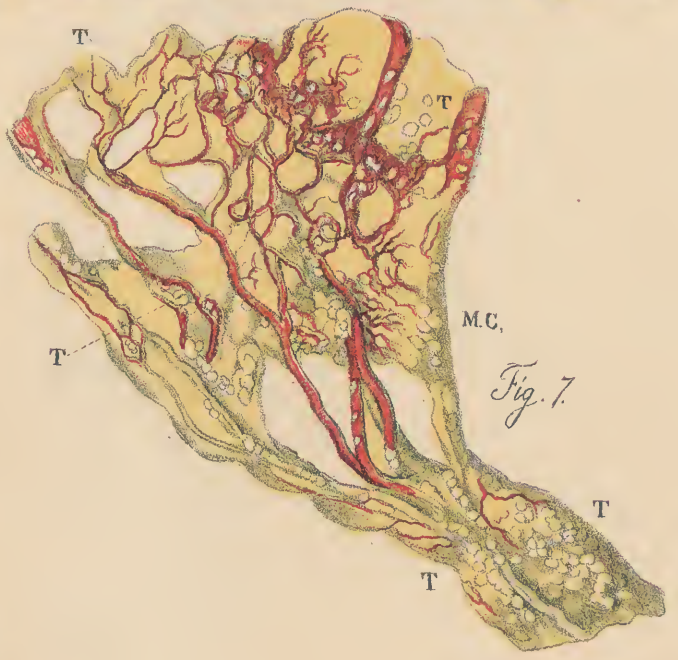

Fig. 7.

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DISEASES OF THE CEREBRO-SPINAL AXIS AND ITS MEMBRANES.

SUB-ARACHNOIDAL MENINGITIS OF THE CONVEX SURFACE OF THE BRAIN (COMATOSE FORM).

Section I, Table I.

Case. — Leclerc, a powerfully-built carriage-driver, fifty-three years old, is taken with erysipelas, limited to the inner surface of the right arm, on the 24th of February, 18—. The surrounding tissues are red and œdematous; complains of pain and heat in the right lower extremity; the pulse is large and frequent.

25th. Patient seems drowsy; other symptoms unchanged.

26th. Stupor; supine position, immovable. Tongue natural, no pain anywhere; abdomen soft; no vomiting. Small, frequent pulse; respiration hurried. Slight delirium in the night, also light spasms of extremities.

27th. Lies prostrate; excessive weakness in limbs. Answers questions sensibly, though very slowly, and in a snappish manner. All organs of sense seemingly sound. No loss of sensation or motion; no particular symptoms of chest or abdomen; pulse 58, soft.

28th of Feb., 1st and 2nd of Mar. Torpor of the body increasing steadily; drowsy, partly comatose, hard to waken. Masticatory motion of the jaws.

Mar. 3d. The eyelids closed and glued together by pus; the iris still sensitive. Complete loss of sensibility of the skin.

4th. Coma.

5th. Stertorous breathing.

6th. Death.

Post-mortem Autopsy. — Fore-arm infiltrated to a portion of its extent with pus. Slight œdema of the right thigh and leg. Thoracic and abdominal viscera perfectly sound. The skull opened: convex surface of brain (Table I, Fig. 1) presents a semi-solid mass, mainly situated in the sub-arachnoidal cellular tissue, and extending to the basilar surface of the hemispheres, covering them. It is not very uniform in thickness; thickest along the course of the vessels which it follows and envelopes; passes between the convolutions and constitutes a pseudo-membraneous layer corresponding with the thickness of the sub-arachnoidal cellular tissue. The arachnoid facing the dura mater is intact.

Symptoms and Pathology. — A state of torpor and extreme sensation of lassitude at first characterizes this form of meningitis. A gradual prostration and immobility follows. Involuntary discharges from intestines and kidneys generally exist; though they may sometimes not take place. Death is caused by asphyxia, from paralysis of the respiratory centra. The chief symptoms seem to be gradual reduction of the forces of the body and steady loss of mental energy, which may last for quite a while, but which will certainly terminate fatally, when the slightest injury or inflammation affects the body.

Explanation of Table I.

Figs. 1 and 3 show the convex surface of the cerebral hemisphere. The vessels, especially the veins, are turgid. The dura mater is of a slightly yellowish tinge, showing through the yellow layer of the sub-arachnoidal pseudo-membrane. Its fibrous structure appears partly infiltrated, and has a slightly opaline lustre. No trace of œdema perceptible in the arachnoidal space.

Fig. 3. A portion of the right hemisphere of another individual affected with meningitis with a purulent pseudo-membrane in the sub-arachnoidal space. Only one hemisphere was attacked in this case. There was not only much clotted pus, but also purulent flocculi floating in large quantities of serum below the membrane. Divested of its membranes, the brain, on a level with the inflamed space, showed very many red spots, of different sizes, both upon its surface and in the gray substance. There was a very copious effusion of serum between the membranes and the brain, all over both hemispheres. Within the affected hemisphere no trace of apoplexy could be distinguished, while within the opposite one, clear marks of an apoplectic focus were visible.

(N. B. — Sub-arachnoidal meningitis may be confined to only one hemisphere, or even to a portion of it, without manifesting any particular symptoms on one side more than on the other.) In this case, the inflammatory process of the membrane did not continue into the apoplectic hemisphere, which seemed to have had no connection with the present cerebral lesion.

Fig. 2 shows the sub-arachnoidal cellular tissue to be pervaded by a yellowish-white clot, extending along the vessels and the sulci of the basilar surface of the brain, and investing the choroid-plexuses.

Case. — A young woman, twenty-one years old, was taken to the hospital (Maison Royale de Sante) in a very disorderly state. No previous history given.

Symptoms. — Questions asked she answers in a very short, sharp way — in a whimsical and conceited manner. No vomiting, nor headache. Face is pale and bears an expression of surprise; pulse is almost natural. Respiration irregular, sometimes very frequent, sometimes very slow. She is not willing to undergo medical treatment. Next day seemingly very much better, yet there was something strange in her manner and in the expression of her face and attitude of her body, indicating the presence of effusion in the cerebral ventricles and sub-arachnoidal cellular tissues of the base. The following night violent, inarticulate screams for several hours.

3d day. Face much altered, and the whole body exceedingly restless. To the questions asked she gives sometimes proper answers, sometimes confused ones.

4th day. Very perceptible wasting of the body, and exceeding change of the features of the face; more restless than ever, more violent in behavior.

5th day. Loss of consciousness; involuntary discharge of urine; strabismus; pupils unequally dilated; pulse very small and exceedingly slow; respiration sighing.

Dies on the tenth day.

Post-mortem. — The body wasted and exceedingly changed. The whole of the left pleura covered with recent pseudo-membraneous deposits; the left lung collapsed and partly splenified; no serum in the pleural cavity. There was some serum in the cerebral ventricles, but no alteration of their walls. In this case there was no headache or vomiting. As a general thing, in this class of cases vomiting constitutes one of the prodromal symptoms, which may continue and become the most prominent one during the whole duration of sickness. Usually, sub-arachnoidal meningitis has not the intense characteristic headaches marking arachnitis proper in its train; it is more dull and stupefying, with here and there shooting pains, which cause the patient to suddenly scream. In some cases, connected with acute hydrops of the cerebral ventricles, the vomiting is so intense and persistent as to simulate softening of the coat of the stomach; but the injected condition of the conjunctiva and dilatation of the pupils will at once indicate that there is compression of the origin of the optic and the ciliary nerves, or serious effusion or pus in the membranes or cavities of the ventricles.

Fig. 4.—*Sub-arachnoidal meningitis of the brain.*

Case. — Letouze, twenty-nine years old, brought to the hospital in a state of stupor. The face has an expression of surprise. Answers no questions. The skin is enormously sensitive. When touched the patient screams and wrinkles the forehead.

History. — Patient was given to all sorts of excesses; used to complain of habitual headache. Was five days ill before entering the hospital. The day after, complete anæsthesia of skin. Involuntary discharge of urine.

3d day. The eyes half open and turned up. The right conjunctiva highly injected. Breathing very changeable.

4th day. Same condition.

5th day. Breathing short, stertorous, gasping. Eyes fixed, and exceedingly injected. Died the seventh day.

Post-mortem Autopsy. — The cervical ventricles contain a small quantity of serum. The fornix is sound, also the ventricular walls. The vessels of the pia mater are much injected. Light infiltration of the sub-arachnoidal cellular tissue of the fornix, which is readily removed. Considerable infiltration of serum in the cellular tissue of the base. It is thick, and semi-transparent.

FIG. 5. — *Sub-arachnoidal meningitis with deposits of concrete masses of pus and tubercles.*

CASE.—A young child, six years old. Appearance of face natural; clear skin; small and uneven pulse. Natural respiration. Answers all questions asked. Complains neither of headache nor of other pain in the body, nor is he drowsy. Has been sick about a day and a half, and vomited constantly during all that time. Does not feel the mustard draughts applied to the abdomen. Remains three days in the same condition.

4th day. After an apparently long sleep, still seems very drowsy, and answers, very slowly, questions asked him; sometimes not at all.

5th day. The right eye wide open, the left closed by the paralyzed lid; both pupils much dilated. Sensibility almost extinct in the left upper limb, but not in the right. Skin still natural, and pulse slow.

6th day. Pulse more frequent, and skin slightly warmer. Answers slowly some questions. In the evening of the same day, an exceedingly strong paroxysm of fever. A very red face, a burning skin. Dies the same night.

Post-mortem. — Skull opened. The pia mater very much injected. The brain covered with an enormous number of vascular loops; no serous effusion below the arachnoid, the pia adhering tightly to the brain. A few loose filaments between the layers of the arachnoid investing the dura and the pia. At one point there was strong adhesion between the two. A tubercular mass at this point. The basal surface (Fig. 6) presented the same injected appearance. The sub-arachnoidal cellular tissue infiltrated with serum, and invested with a brownish pseudo-membrane. The arachnoid much thickened. Within the Fissure of Sylvius, a large quantity of whitish tubercles (*F.*), of different sizes, attached to the vessels and the cellular tissue; a small amount of serum in the ventricles. The fornix reduced to a pulp.

Fig. 6 shows only one hemisphere. The middle lobe removed to show the Fissure of Sylvius in its whole extent. Section of the middle lobe (*L. M.*).

Fig. 7 represents the meningiæ detached and spread out, showing a considerable number of tubercular masses (*T.*) within the cerebral substance, and along the vascular windings (*T. T.*).

Fig. 8 shows the cerebral substance (*S. C.*), from which the membranes were carefully detached, covered with tubercles, which followed, in their formation, the tract of the membrane from the cellular tissue, and imbedded themselves in the substance of the cerebral cortex. These tubercles having become a source of great irritation, caused a softening of the substance of the brain, and are here shown as red points of different sizes. Most of these tubercles were hollow.

In this case, where apparently only a slight injury to the brain existed, the cause of sudden death seems due to the acute formation of the tubercular masses, and the diffuse inflammation of the cellular tissue extending into the brain.

TABLE II.

Hemiplegia, with excessive pain in the limbs and epileptiform spasms. Two cancerous tumors of the dura mater on the convex surface of the brain. Gelatiniform softening of the cerebral substance.

History of the Case. — Name of patient, LECOUVREUR; sixty-five years old; affected with hemiplegia on the right side, except the face. In full possession of his mind. Suffered excruciating pains in the whole body, and was attacked occasionally with epileptiform spasms. During the spasms, the lame side, as well as the healthy side, was convulsed. The slightest movement made by the lame side produced enormous pain. Pulse nearly normal. Took some nourishment. Bed-sores from constant position on the back. Died, after long agony, in full possession of his mental faculties.

Post-mortem. — On top of the skull, in the region of the sagital suture, near the fronto-parietal (*S. F. P.*), a considerable prominence was seen, distinctly visible during life. Beneath a very fibrous layer, very adhering and very vascular, a quantity of bony vegetations (*V. O.*— Fig. 2) was found. These vegetations, forming a spongy tumor, were held to the skull by means of the soft parts, their base facing the inner surface of the skull, which they had perforated (*P.*), and their apex upon the dura. The tumor could readily be compressed, but soon rebounded like an elastic ball. When the skull was removed, it was found that the internal surface was much corroded in a very irregular manner. A reddish carcinomatous substance filled up the uneven spaces, and extended into the Diploe. The debris of the destroyed bone and the carcinomatous substance adhered to the (*D. O.*— Fig. 3) dura mater above and by the side of the superior longitudinal sinus. The opened sinus presented small vegetations, which spread upon its walls and completely closed it up. Removal of the dura showed (Fig. 1) two spheroidal carcinomatous tumors (*Te. Te.*) of unequal size, having their origin on the dura mater in the receding angle formed by the Falx cerebri (*F. C.*) with the dura mater (*D. M.*). The tumors were not formed on the brain, but they caused a deep depression upon its surface, and did not adhere to it. The arachnoid and pia mater were completely grown together with the tumor. The convolutions of the brain were obliterated where the tumors pressed upon them. They were invested with many blood-vessels of different caliber. A vertical section of the left hemisphere, antero-posterior to the largest diameter of the larger tumor, was made, and here represented. Beneath it (Fig. 3) the convolutions were obliterated, and only a thin layer of gray substance remained. The surrounding cerebral substance (*S. C.*) was exceedingly softened and semi-transparent, resembling a trembling mass of jelly (*gelatiniform softening*), of yellowish-white color. Very tortuous veins of larger caliber than usual penetrated this jelly-like mass, and passed into the Corpus Callosum. The section of the tumor (Fig. 3) presents soft encephaloid and tubercular masses, traversed by a great many vessels, forming radiating groups or clusters.

Symptoms and Pathology. — The hemiplegia existing during life was evidently the result of the compression exerted by the larger of the two tumors upon the left hemisphere. The hemiplegia was to the right. The irritation caused by this pressure was the cause of the exceeding pain. (N. B.—There are sensory centra in the cortex of the brain.) The patient never suffered headache, but all the pain was felt in the periphery of the body. The tumors formed upon the inner surface of the dura, facing the brain, which it caused to atrophy by pressure. These, primitively a mere source of compression, gradually become a source of irritation, causing on one hand hemiplegia, on the other, excessive pain. At first confined to the internal surface of the dura, the cancerous degeneration continued through its thickness, then to the bony plate and to the marrow cavity, and perforated by its corrosive action the outer plate.

Fig. 2.

Fig. 4.

Fig. 3.

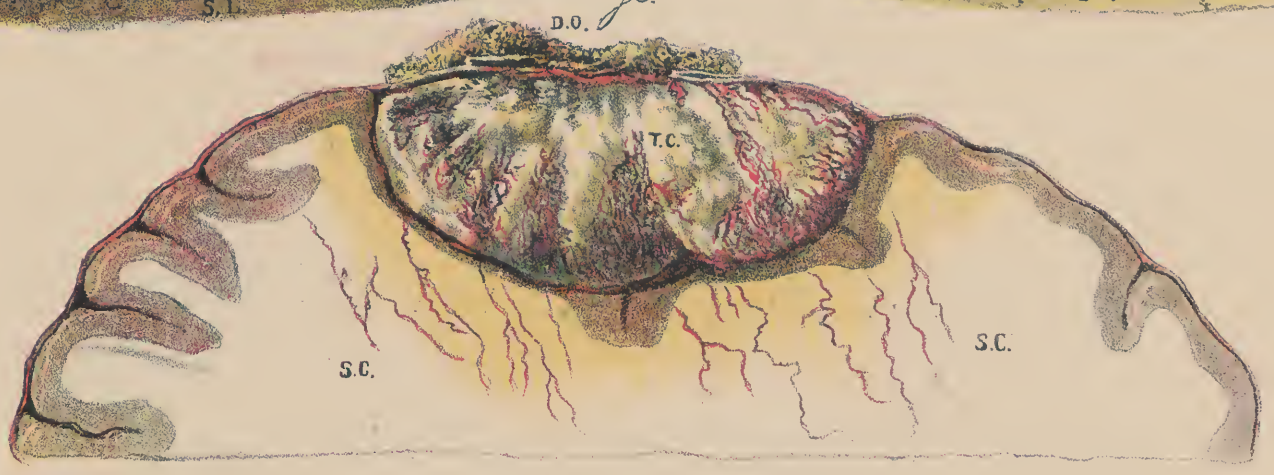
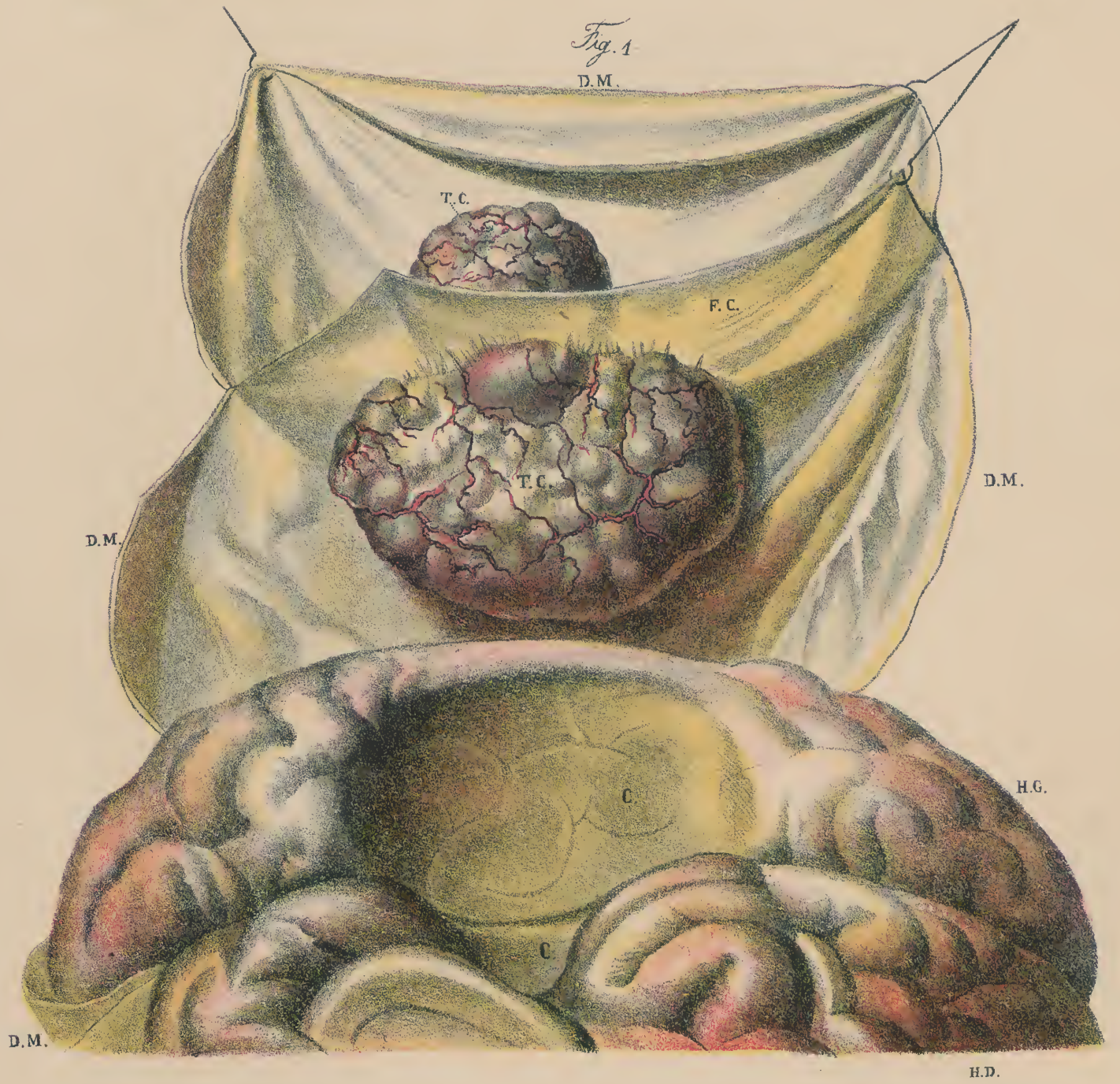


Fig. 1



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TUMORS ON THE FALX CEREBRI.

Sec. 1. *Tab. III.*

Fig. 2.

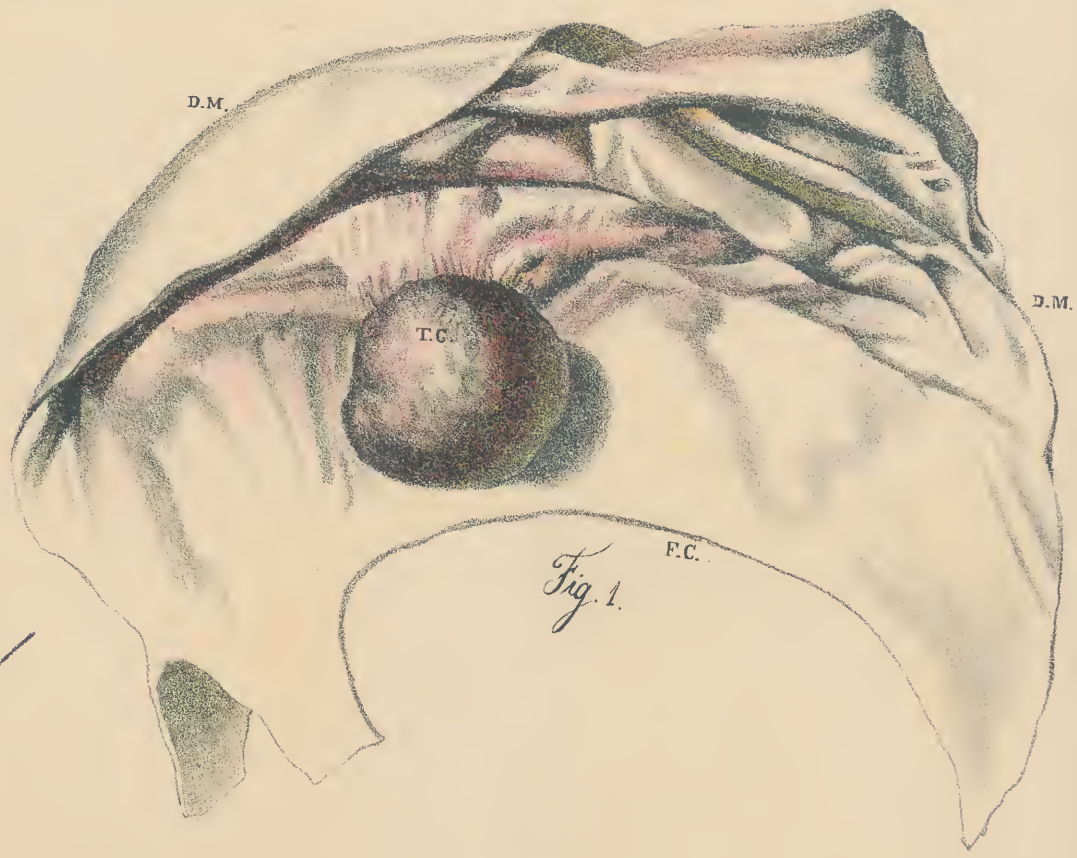
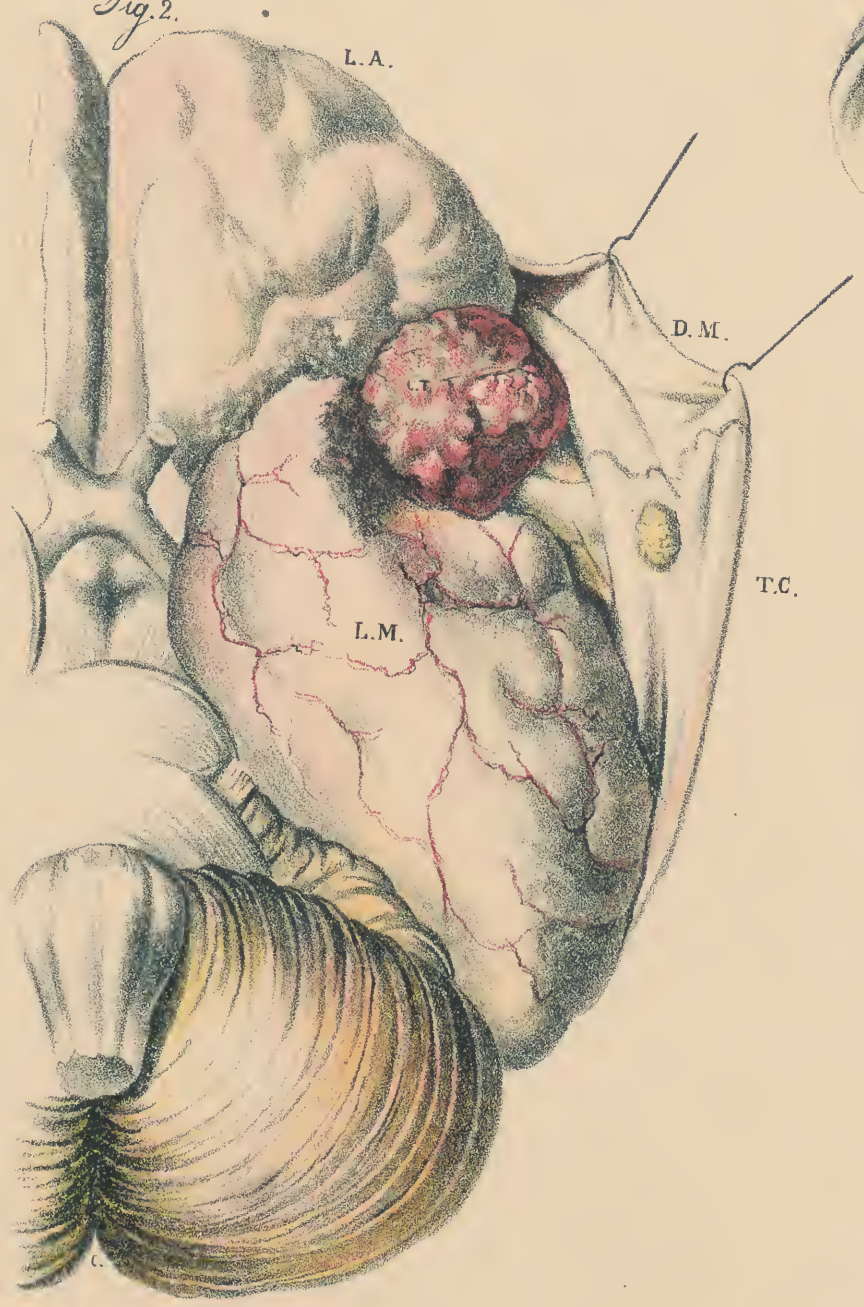


Fig. 1.

Fig. 2.



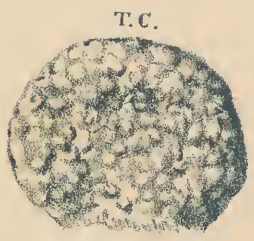


Fig. 1'

Fig. 3.

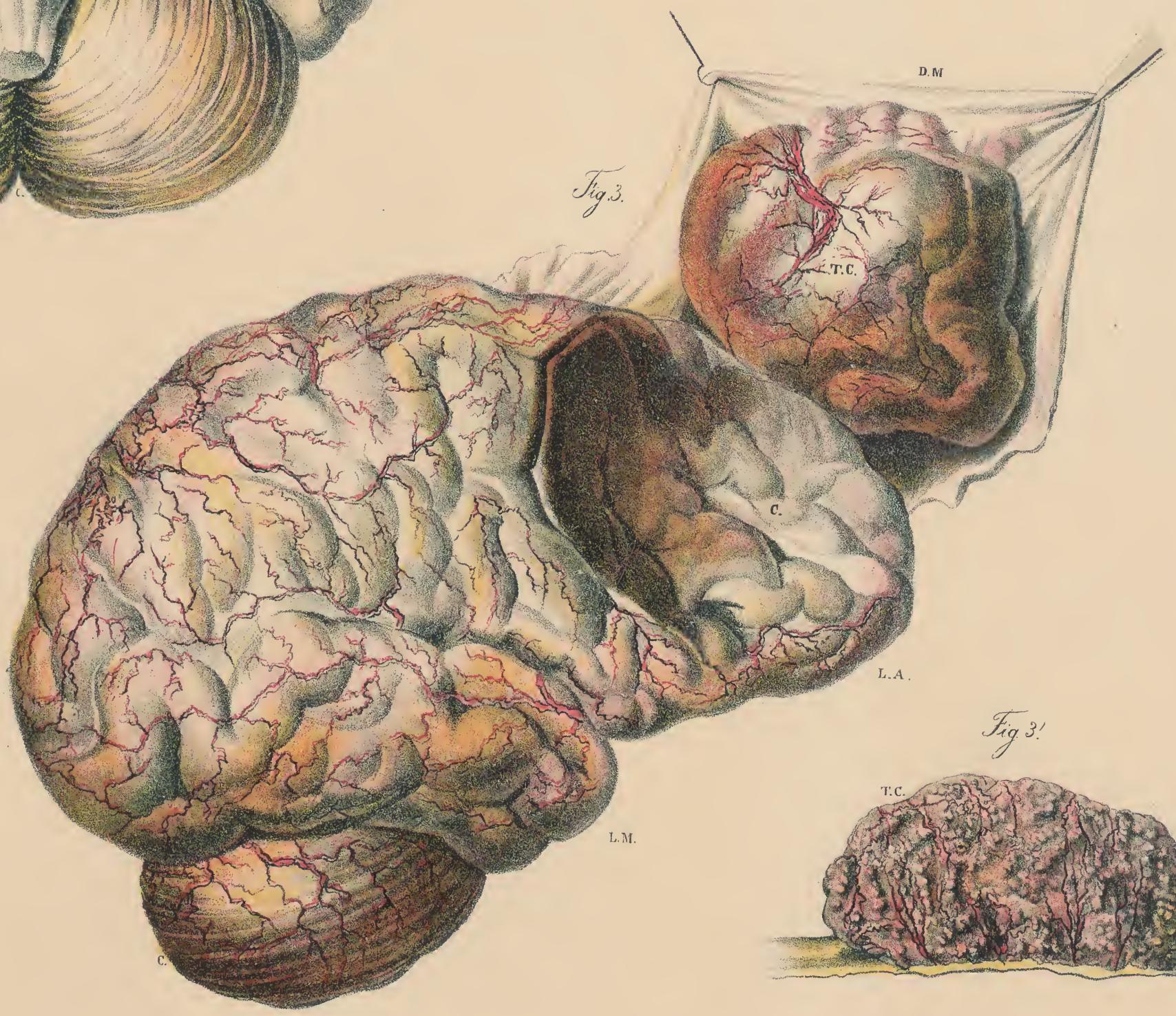


Fig. 3'

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When carcinomatous tumors develop upon the outer surface of the dura, they spread outward, perforate the skull, and grow outside. But when they develop upon the inner surface, they extend both inwardly and outwardly, and exert chiefly their injurious effect upon the brain (*Cruveillhier*). The very dense and close cellular tissue, uniting the cervical layer of the arachnoid with the internal of the dura, seems to be the most frequent seat of tumors of this kind.

TABLE III.

Carcinomatous tumors formed upon the Falx cerebri.

Fig. 1 shows a spheroidal fibrous tumor (*T. C.*), formed upon the right lateral surface of the Falx cerebri (*F. C.*), near its convex border. The tumor was lodged in the depth of the right cerebral hemisphere, which, however, it left uninjured, as well as its investing membranes. It was of almost cartilaginous density, smooth on its surface. Cut with the scalpel, it emitted a crackling sound. It was torn with great difficulty, and presented on its cut surface a tubulated appearance. It seemed of a fibrous tissue, and impregnated with a milky liquid, which was easily pressed out, and apparently was of a scirrhus nature.

CASE.—MR. R., aged sixty-five years, suffered with stone in the bladder; was operated on by lithotripsy, but without success. Consulted Leroy d'Etiole and Dupuytren, who advised against lithotomy. Was affected for two years with paralysis from brain lesion, and subsequent œdema of the lower limbs. The whole body was bent forward when in a sitting posture. He could not get up; when raised up and placed on his feet, he could walk a little by dragging them, but could not lift them. His mind was perfectly clear. Was slightly deaf in both ears. Eventually Mr. Dupuytren operated on him for lithotomy; extracted two calculi, one very large. Died shortly afterward.

Post-mortem Autopsy.—The body very speedily, after death, underwent decomposition. On opening the skull a notable quantity of serum ran out. The brain was healthy, and of natural consistency. Cutting down upon the falx cerebri, at its anterior extremity, and turning it over, a fibrous tumor of about an inch and a half in diameter, situated on the right surface of the falx, and lodged on the internal surface of the right hemisphere, was found. The brain as well as the arachnoid in that locality were perfectly sound. Thorax and abdomen normal. The kidneys enlarged, and very much softened. The bladder contained in its *trigone* a third calculus, of the size of a hen's egg. A number of pockets, containing calculous deposits, were also found.

Fig. 2 shows a spheroidal tumor (*T. C.*) on the dura mater, on a level with the Fissure of Sylvius, and penetrating into the anterior extremity of the middle cerebral lobe (*L. M.*). A smaller tumor (*I. C.*) visible behind and outside the former. It was of a carcinomatus nature in its incipient stage. The figure represents a vertical section of the larger tumor. It was filled with a yellowish viscid substance. A large collection of serum in the sub-arachnoidal cellular tissue, and in the cervical ventricles, seems to have caused the death of the patient, by producing compression of the brain.

Fig. 3 shows a carcinomatous tumor on the external surface of the brain.

CASE.—A female school teacher, aged forty-five, was afflicted with habitual headache. Entered Maison Royal de Sante Hospital, Paris, France, September 3, 18—.

Symptoms.—Permanent supination of body. Motionless; sometimes drowsy. Involuntary discharge of urine. Bowels constipated. Asks for nothing, but also refuses nothing. No local paralysis of either motion or sensation. Was made to raise up, supported by two waiters, and requested to try to walk, but though apparently willing to do so, was unable to move forward, her limbs bending under her weight. Could barely hold up her head. The left corner of the mouth was somewhat depressed. The questions asked she answered in a very low voice, and very slowly. Seemed to experience difficulty in speaking. When asked where she felt pain, she carried her hand to her head, in the frontal region. For several days she was helped to a seat in an easy chair, which seemed to be agreeable to her. Would still keep up in a half-lying-down, supine position. Her pulse weak, and very variable as to frequency and volume. After a fortnight, could not rise any more, and could not even sit up, from lack of muscular power. The intellect undisturbed. The action of the pulse, very frequent and very feverish. Died the same day.

Post-mortem Autopsy.—Removal of the top of the skull, and carefully throwing back the dura mater, a very voluminous tumor (*T. C.*—Fig. 3), springing from the inner surface of the dura mater to the right of the falx cerebri, was shown. It was lodged in a cavity formed by the tumor, within the anterior lobe of the right cerebral hemisphere.

The convolutions were compressed and atrophied, but not obliterated. The tumor was readily enucleated from its seat. Its surface was uneven, and completely covered with vascular net-works. It was softer than the cerebral substance, and very readily compressed. The figure is an exact representation of its aspect. Innumerable granulations were scattered within its soft interior pulp, and lodged within vascular meshes. These vessels were continuous with those of the dura mater.

Symptoms and Pathology.—The only prominent external symptoms were, a general torpor without any notable diminution of the intellectual faculties, which required but a slight effort to set to work; a general weakening of motor power, rather more to the left than to the right. The peculiar position of the tumor readily indicates the reason why, in this case, there was *general paresis*, though the tumor seemed to exist on one side. A portion of the right as well as the left hemisphere was compressed. That the intellectual faculties were but slightly affected, was owing to the uninjured state of the convolutions, though they were somewhat compressed. (*Cruveillhier, Anatomie Pathologique.*)

TABLE IV.

Acute sub-arachnoidal meningitis of the whole surface of the cerebrum and cerebellum. Delirium the first two days. Coma and death the third day. (Martin, pathological specimens presented to the Societie Anatomique.)

CASE.—NETEVAL, seventy years old; blind. Entered Bicetre Hospital Feb. 16, 18—.

Symptoms.—Cheeks very red; talked incessantly; is sometimes furious. The questions asked he answered with oaths and imprecations. He is exceedingly restless in bed, and throws himself right and left; tries to get up and go away. The sensitiveness of the skin is undiminished. Does not complain of any pain. The pulse exceedingly frequent, full, and hard. Temporal arteries beat very violently. The head hot and dry. Next day, movements less violent; still talks very much, and does not finish his sentences. Pulse weaker. Light slumber.

3d day. Deep coma; face discolored; respiration slightly stertorous and sighing. Skin perfectly insensible to pain. Upper limbs rigid, in flexed position; slightly tremulous. (He was very much given to strong drink.) Died on the third day.

Post-mortem Autopsy. — The brain (Fig. 1, Table 4) is enveloped all over by a greenish-yellow layer of great density. At first sight the layer seemed to be within the arachnoidal cavity, but careful examination showed it to be a purulent pseudo-membrane contained within the space between the arachnoid and the pia mater, the vessels of which, highly developed, were distinctly visible through this half-transparent infiltrated mass. It was thickest along the tracks of the vascular trunks. Not only did it cover the brain, but it also penetrated into all its anfractuosities, with the sub-arachnoidal cellular tissue (*A. A. A.*). This is plainly visible on the right hemisphere (*H. D.*), from which the internal third and the corpus callosum were removed, and the two outer thirds turned outwardly. The vessels (seemingly veins) which are well developed, belong to the pia mater, and contrast with the whitish-green color of these prolongations, which are very thick, and, consequently, compressed the convolutions. The basilar surface of the brain presented the same appearance as the convex. The pseudo-membraneous mass was most abundant in the midian basilar cavity and along the cellular prolongations, which seemed to protrude from it. A similar mass filled up the space between the arachnoid and the pia mater of the cerebellum (*C.*). It is also extended to the choroid plexuses (*T. C.*). The ventricular cavities presented no trace of inflammation, and the cerebral substance seemed unaltered. The patient suffered from chronic alcoholism.

Figs. 2, 3. *Inflammation of the superior longitudinal sinus.*

CASE.—Entered March 17, 18—. A child four years old; affected with enlargement of the lymphatic glands, and ophthalmia.

Symptoms.—Dilated pupils; sighs and moans; tongue white; has vomited several days previous eight Ascarides lumbricoides.

18th. Pupils still dilated. Screams. Limbs rigid; can not hold up the head. The tongue protrudes from the mouth from a tumor within the sub-maxillary gland. Inflammation of the apex of the left lung discovered.

21st. Perfectly calm, but comatose.

Died on the 22d.

Post-mortem Autopsy. —A great amount of serum in the arachnoidal cavity. The superior longitudinal sinus (*S. S.*) is filled with coagulated blood, strongly adherent to the walls of the sinus. In the center of these clots a half-concrete purulent mass is seen. This did not come in contact with the vascular walls. All veins emptying into this sinus are full of semi-solid pus, which form yellowish-white strings, separated from each other by clots of blood. A few of the veins (*V. P. U.*) are opened to expose the bloody and purulent cylinders.

Fig. 3. The Torcular Herophili is also laid open, showing its distention with the sanguo-purulent masses. All other sinuses were unaffected. A sort of ecchymosis is visible on the left hemisphere, near the superior longitudinal sinus.

GENERAL PATHOLOGY OF THE NERVOUS CENTRA.

The nerve centra are composed of ganglia or superposed cellular groups, and of nerve fibres which unite them.

In the spinal marrow these ganglia form a continuous chain, with two enlargements, one brachial and the other lumbar. The ganglionic chain comprises two cellular columns, an anterior and a posterior.

The first (anterior cornu) is made up of large cells, communicating with the motor roots. An acute or chronic inflammation can affect its whole length, and determine cellular atrophy. It gives rise to certain diseases known by clinical names, as *infantile paralysis, spinal paralysis of the adult,* when in an acute form, or as *progressive muscular atrophy* of Duchenne, when the disease is in a chronic form.

This cellular motor column may also be affected secondarily, through the spread of diseases of the neighboring tissue, and thus produce a complication with the primary disease, that is muscular atrophy and loss of reflex action.

The posterior column is composed of smaller cells, which occupy the internal portion of the posterior cornu. It is also called the *vesicular column of Clark.* According to the most recent researches of Pierret, it comprises three principal groups: a dorsal, a cervical, and a cranial. The last constitutes the nuclei of the *Trigeminus,* in the Medulla Oblongata.

The nerve fibres of the spinal marrow are of three kinds:

1. The fibres of the anterior and posterior roots, which cross the spinal marrow horizontally, from before backward, or from behind forward. An inflammation of the anterior cornu extends very often to the nerves and the corresponding muscles, in following the track of the roots. In the posterior roots, an inflammation extends, as a general rule, in an ascending direction.

2. The longitudinal commissural fibres which unite the superior ganglia with the inferior and transverse commissural fibres, which bring the two halves of the spinal marrow into communication.

3. The cerebro-spinal and spinal fibres properly speaking. The first are of two kinds, anterior or motor, and posterior or sensory.

When the cerebro-spinal motor fibres are systematically affected, that is, when sclerosis is developed longitudinally, paralysis of voluntary movements will ensue. This paralysis is often transformed into contractions of the muscles, by the progression of the disease toward the neighboring fascicles. They constitute the *descending degeneracy* consequent upon lesions of the cerebral hemispheres.

When chronic inflammation affects the spinal fibres proper, there will follow functional spasms, contractions and muscular paralysis, and often atrophy. Charcot describes this disease as a lateral *Amyotropic Sclerosis.*

When chronic inflammation spreads into the posterior cords, terrible pains, neuralgia, gastric troubles, and motor ataxy will be the consequence. They constitute the *Atactic Tabes Dorsalis.*

Transverse inflammation of the spinal marrow (whether spontaneously developed or produced by slow compression — as it happens in Pott's disease — by tumors, or by diffused spinal Pachy meningitis) determines a complexity of symptoms, resulting from a combination of all the preceding.

In the medulla oblongata the nerve ganglia are distributed in the floor of the ventricular gray matter. They constitute what is ordinarily called the nuclear origins of the cranial nerves.

These ganglia may become the seat of sub-acute or chronic inflammation, producing the constant clinical syndrom: that is, *labio-glosso-laryngeal paralysis.*

Diseases of the medullary cords may also spread into the corresponding white bundles of fibres of the Medulla Oblongata; then there will be super-added the following symptoms: lateral sclerosis, labio-glosso-laryngeal paralysis, and atrophy.

In posterior sclerosis, terrible pains and ataxy of the face and eyes exist.

In the cerebral hemispheres, the following pathological localization has been established: In the white substance, on the same plane with the peduncular expansions, complete hemiplegia follows a lesion of its anterior portion.

Hemianæsthesia, both of the organs of special sense and of common sensation, follows a destruction of its posterior portion.

Hemichorea and athetosis follow diseases of the most posterior nerve fibres of the *Optic Thalamus.*

"In each cortex cerebri, a large motor zone has been discovered. A lesion of the whole zone produces extensive paralysis of the whole opposite half of the body; partial lesions are followed by local motor troubles, such as paralysis of the face, one limb, a certain muscular group, etc., or partial epilepsy." (*Duret, Etude Generale.*)

Cerebral hemiplegia having a central origin (according to Chorcat, Pitre, Duret, Ferrier, etc.).

The clinical character of this kind of hemiplegia is as follows: 1. It constitutes a paralysis of motion of the whole side opposite to the diseased centrum. 2. It is intense, definite, and incurable. 3. It is always followed by progressive degeneracy of the lamed side, which ends in contraction. It differs from hemiplegia of a cortical origin in this, that the latter is never so extensive nor so complete, nor are the tissues of the lame side so flabby; and is generally followed by a speedy recovery. The most frequent cause of the lesion is a hemorrhagic focus, differing in extent, but constant in its locality. It is situated in the anterior portion of the internal capsule, between the caudate and the lenticular nuclei. Such a focus can very readily be studied, and all its anatomical relations analyzed, on a transverse section of the hemisphere made a few milimeters in front of the chiasma of the optic nerve. It is due to a rupture of one of the small arteries, designated as the lenticulo-striated. These vessels have their origin a short distance from the origin, of the Sylvian artery, on a

DISEASES OF THE CEREBRO-SPINAL AXIS

CEREBRAL MENINGITIS. INFLAMMATION OF SINUS.

Sec. 1. *Tab. IV.*

Fig. 1.

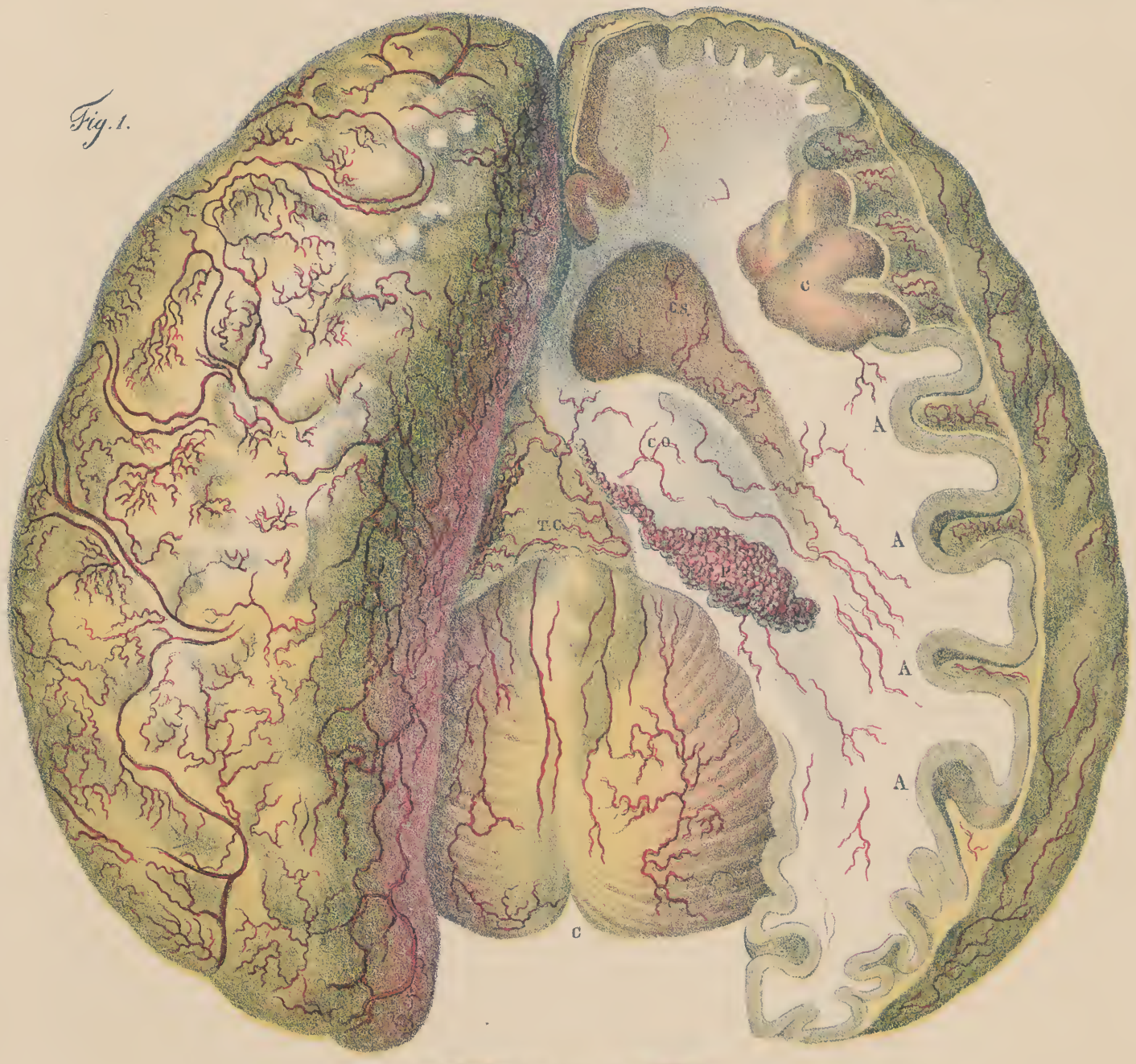
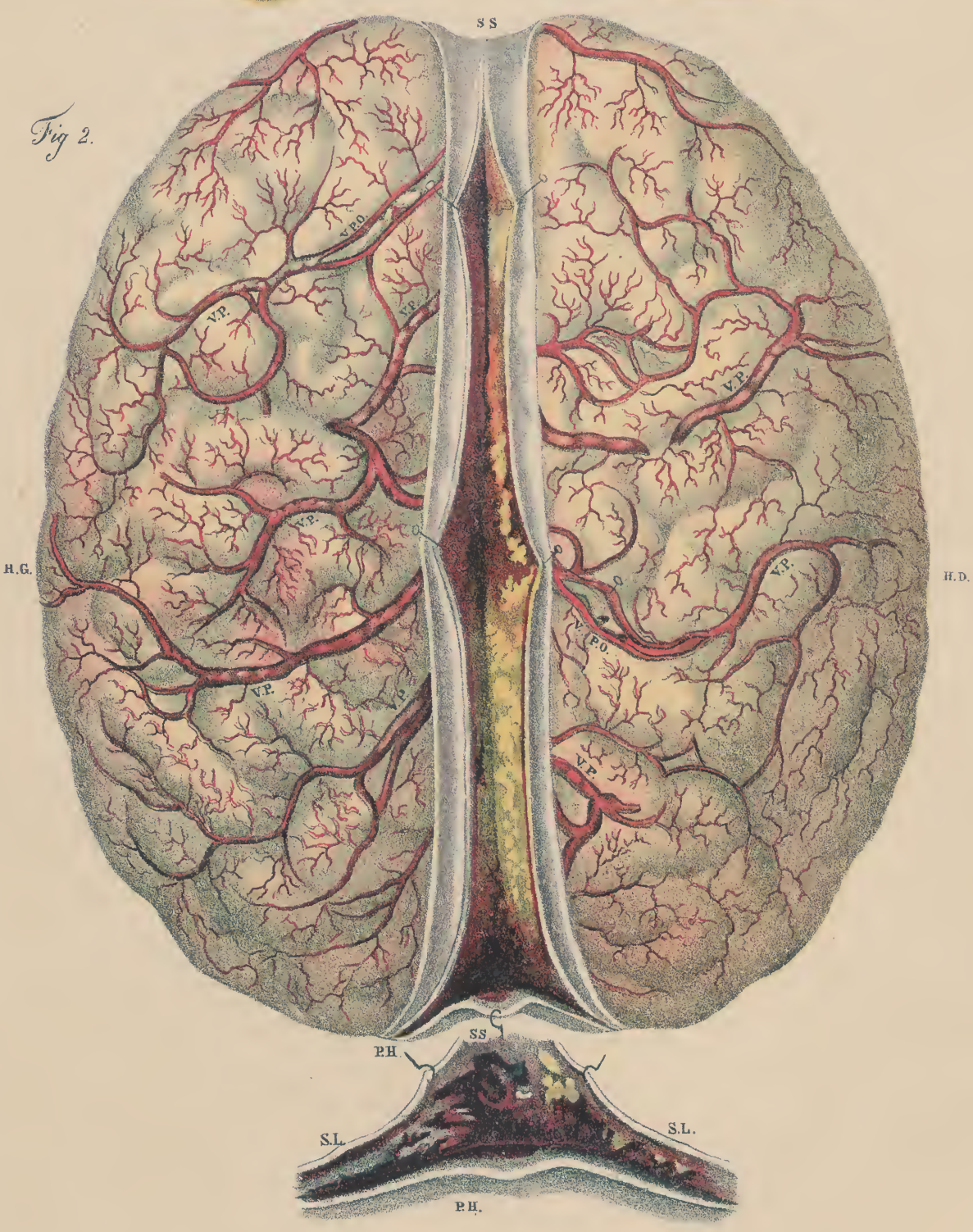


Fig 2.



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PACHYMENINGITIS.—LEPTOMENINGITIS.

Sec. I. *Tab. V.*

Fig. 2.

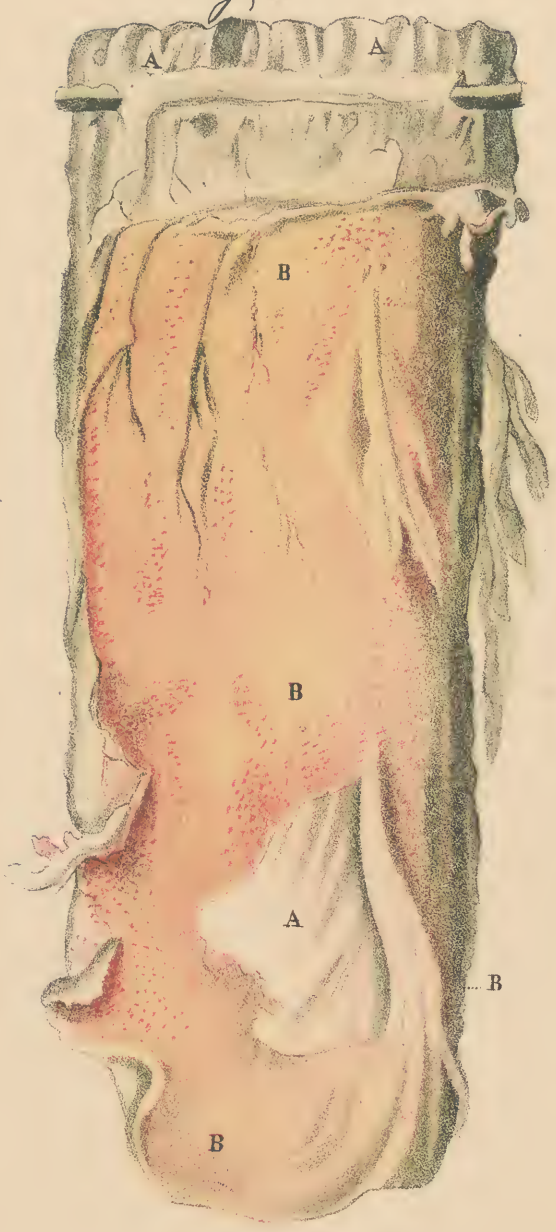

Fig. 1.

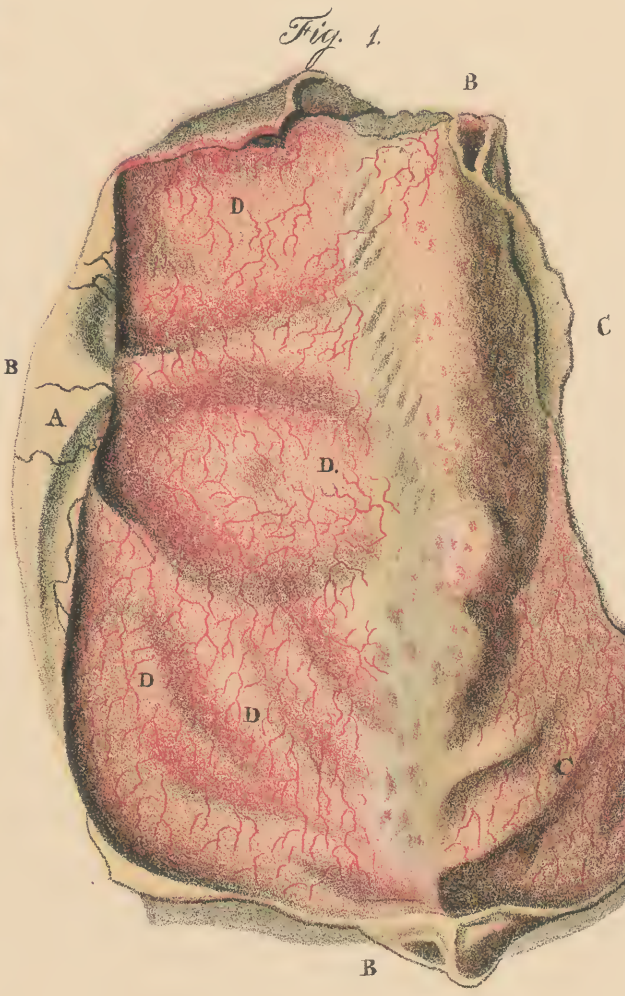

Fig. 3.

Fig. 4

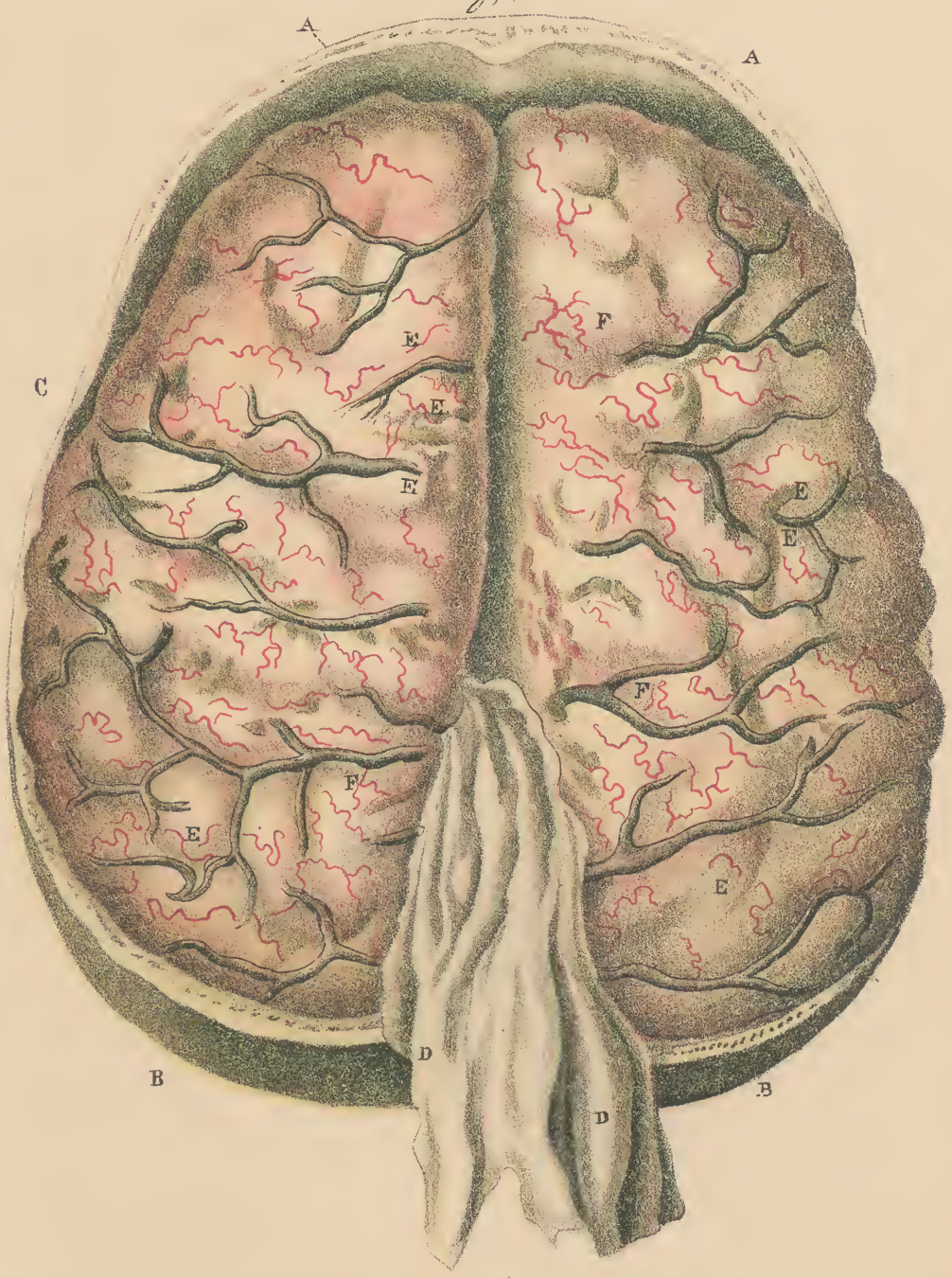

Fig 5

TABLE V.

ACUTE HÆMORRHAGIC PACHYMENINGITIS AND LEPTOMENINGITIS.

FIGS. 1, 2 AND 3.—*Acute inflammation of the Dura Mater, with exudation and subsequent adhesion to the other membranes.*

In by far the greatest number of cases the inflammatory process takes place in the region of supply of the middle meningeal arteries, and spreads thence behind and in front.

The hyperæmia manifests itself by dilatation of the arteries, which become thereby more tortuous than usual, and surrounded with a spongy *adventitia*. To the internal surface of the membrane, which is usually white and smooth, it gives a light rosy color, by over-filling the vast number of exceedingly fine capillaries. This color remains during the whole stage of the process. Upon this so slightly changed surface a loose semi-transparent, yellowish layer of variable thickness is formed. Scattered over this are great numbers of smaller or larger red spots. The layer is very readily detached from its basis by forceps. By removing it carefully a number of fine and coarse strings, which attached the pseudo-membranous layer to the surface of the dura, can be noticed to have remained behind.

These strings (or, really, newly-formed vessels) are derived from the dural surface and pass into the neoplastic membrane, and are distributed through it in a radiate manner. The membrane itself will be found, on examination, to consist of vast vascular networks (the walls of these vessels are very incompletely organized as yet) imbedded in a loose homogeneous, mucus-like substance, constituting an intercellular stratum for some scattered, stellated or spindle-shaped cells. The cells anastomose with one another by their processes.

The whole resembles neoplastic membranes formed upon serous structures, generally.

Fig. 1 represents a portion of the inflamed dura mater covering the right cerebral hemisphere. The internal surface shows the vascularized condition, produced by inflammation.

A. External surface. *B.* Longitudinal sinus.
C. Falx cerebri. *D.* Internal surface.

Fig. 2. A portion of the dura mater, of another individual affected likewise with hæmorrhagic pachymeningitis, in a more advanced state of inflammation.

The neomembrane is formed and partly organized.

A. Internal surface of the dura.

B. The newly-organized membrane, into which the vessels, derived from the dura, pass. The red points indicate the hæmorrhagic focuses. The color of the vessels are darker than usual, on account of the stagnant blood in them.

Fig. 3. A portion of a dura of the left cerebral hemisphere (of another individual) similiarly affected.

A. Internal dural surface. *B.* A quantity of completely and partly organized substances of the pseudo-membrane detached from the dura. Dense masses of the exudate obscure the vascularization. Only the larger vessels are visible here and there.

C. Filamentous strings or fringes of coagula, containing vessels, detached from the membrane.

Acute Leptomeningitis, with infiltration in the Arachnoid.

Fig. 4 represents the convex surface of both cerebral hemispheres.

The vessels are dilated and gorged with blood. A considerable quantity of albuminous coagula and pus are found here and there, between the layers of the arachnoid and the dura mater. The arachnoid is infiltrated, opaque, and thickened in some spots.

A. Frontal bone. *B.* Occipital. *P.* Left Parietal. *D.* Dura mater detached from the Crista Galli and drawn backward, between the hemispheres.

E. Yellow spots of purulent albumen between the pia and the arachnoid.

F. Pia mater vessels, turgid with blood.

Fig. 5. Basilar surface of the brain (a portion of left hemisphere removed).

The same morbid phenomena as in the former figure, only in a more advanced stage. Fibrinous exudation on the whole basilar surface. *A.* Anterior lobe of right hemisphere. *P.* Anterior lobe of left hemisphere. *C.* Middle cerebral lobe. *D.* Cerebellum. *E.* Olfactory nerves. *F.* Optic nerves. *G.* Internal carotids. *H.* Infundibulum. *I.* The exudate covering the medulla and concealing portions of the basilar artery and the nerves. *K.* Basiliar artery. *L. L.* Exudate between the membranes.

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level with the anterior perforated space, winding round the anterior surface of the lenticular nucleus, ascending into the anterior capsule, then passing the internal capsule to its superior portion, is then distributed to the caudate nucleus. When a rupture of the artery takes place in the external capsule, the focus is then situated between the external surface of the lenticular nucleus and the Island of Reil; the Preclaustrum is then destroyed. Then the result is a complete hemiplegia at first, but which very soon improves by the absorption of the clot, and which eventually may even heal altogether. Such hemiplegiæ recover, because the Psycho-motor fibres occupying the internal capsule are only compressed by the clot, but not destroyed. There are three kinds of hemiplegia, distinctly differing from one another: 1. A cortical hemiplegia. 2. A central, of the internal capsule. 3. A central, of the external capsule.

An embolus lodged within the lenticulo-striate artery may determine a softening of the anterior portion of the internal capsule, which manifests itself by a complete hemiplegia.

Clinical character of Hemianæsthesia, its diagnosis and nature; and the position of its lesions.

After an attack of apoplexy, by which the voluntary movements have more or less been destroyed on the side opposite to the pathological focus, there is a gradual subsidence of the motor-paralysis; but at the same time the sensitiveness of the parts is completely obliterated; the loss of sensation equally exists in the face, the trunk, and the upper and lower limbs. It equally affects the sense of touch, the sensation of pain and of temperature. The sight, the hearing, the smell, are completely destroyed on one side; even the muscular sense is affected, and the patient will bear strong electric contractions without pain.

A post-mortem will show that the cause of this complete loss of sensation is due to a hæmorrhage, or a softening of the posterior portion of the internal capsule, between the Thalamus opticus and the lenticular nucleus.

Raymond has described a case of a woman affected with such a hemianæsthesia, upon whom he held an autopsy, and which completely confirmed the experiments made upon dogs, in the same sense, by Carville, Duret, and Veysiere. Such lesions are the result of rupture or obliteration of an arterial group, which is named the *lenticulo-optic*. These arteries are derived from the Sylvian within the anterior perforated space; pass backward, wind round the lenticular nucleus, pass through the internal capsule, and are lost in the optic thalamus. When the apoplectic focus is small, and takes place in the external capsule, the sensitive fibres are not destroyed. Hemianæsthesia is the only result of the compression of the internal capsule, and, though general, is but slightly marked, and very prone to get better, and even perfectly well. Bundles of sensory fibres from the medulla occupy both sides of the Pons Varolii. There is a junction of the fibres which unite the nucleus of the Trigeminus, the Auditory, and the Gustatory with the Encephalon. The course of these bundles may be traced on the sides of the pedunculi cerebri, which here expand fan-like, and form the posterior portion of the internal capsule. It is necessary to explain why there exists, in lesions of the posterior portion of the internal capsule, both Amblyopia and Anosmia. Graefe has advanced the theory that a limited lesion in the hemisphere produced a lateral homologous Hemiopia; that is, a lesion in the right hemisphere produces a suppression or a blindness of the right field of vision, or *the reverse*, as the case may be.

LOCALIZATIONS.

There are five kinds of amblyopia: 1. Diminution or abolition of the sight in the eye, corresponding with the anæsthetic side; that is, the eye opposite the diseased internal capsule. 2. No visible lesion of the fundus of the eye. 3. Sharpness of the vision diminished one-half or more. 4. General and concentric contraction of the field of vision. 5. Weakening of perception of color, especially violet—sometimes even green, red or orange.

TABLE VI.

FIG 1.—*Hæmorrhagic Pachymeningitis. Hæmatoma on the right side of the middle cerebral lobe.*

CASE.—A woman about 42 years of age, apparently in good health, was taken suddenly with a very violent pain in the head and neck; medical treatment failed to relieve her; and, shortly after the attack, she became numb all over. Besides the pain and numbness no morbid symptoms. Three days later she became furiously delirious, and left sided hemiplegia appeared. No other cerebral symptoms manifested. Died suddenly the next day.

Post-mortem.—The whole brain severely hyperæmic. The whole subarachnoidal space at the pons and medulla oblongata, especially on the right side, was filled with large clots of blood. The membranes formed a sac, also filled with clots of blood and protruded forward toward the middle cerebral lobe. (The specimen was kept for several days in alcohol, which made the bloody tumor turn paler than in the fresh state.)

FIGS. 2, 3 AND 4.—*Tubercular Meningitis, Cortical Tuberculosis.*

The brain represented here was that of a child of seven years, who appeared in perfect health, with the exception of some eruption upon the neck, which his attending physician designated as scrofulous eczema (?)

About six months before his death he manifested symptoms of melancholy, ceased to associate with his playmates, became occasionally very excitable and ill humored. Complained often of headache. Transitory strabismus was often noticed, sometimes on one eye sometimes on the other. His appetite and general health did not seem to fail to any noticeable extent. Shortly before his death there was much loss of vision in the left eye. Occasionally there was light fever, of nights.

Two days before his death he complained of being very tired, remained in bed and appeared drowsy. Was slightly delirious in the night, and started in his sleep. The day before his death he became very delirious, and was attacked with very severe convulsions. Died the following night in a complete state of unconsciousness.

Autopsy.—On opening the cranial cavity, the left hemisphere, immediately beneath the coronal suture, showed a solid tubercle of about an inch in length and half an inch in thickness. The dura mater was attached to the arachnoid, the pia mater and the brain, and seemed to have been in a high state of inflammation, both, where the tubercle existed and adjacent to it. The membranes were much infiltrated and adherent to the tubercular portion of the brain.

Fig. 2 shows a section of the brain, where the tuberculous mass formed in a sulcus, which it had widened, and the walls of which formed a sort of an arch over it. It compressed the adjacent gyri and softened the cerebral structure around it.

A tubercular nodus was also found upon the superior posterior surface of the vermiform process of the cerebellum, of about two inches in length and an inch and a half in thickness. The tissue surrounding it was in a state of red softening (Fig. 3).

Fig. 4. At the posterior border of the right hemisphere of the cerebellum a hard tumor, connecting the inner surface of the dura mater with the lateral sinus, was found. This was situated in a sulcus between the lobules of the cerebellum (Fig. 4*b*).

An other tubercular mass was found between the cerebellar hemispheres, enclosed in portions of the gray and white substance (Fig. 3*a*). It had pushed the laminæ of the gray substance asunder and compressed the subjacent medullary substance into a very thin layer (Fig. 4*a*). All these tubercles were originally formed on the pia mater, but continued on each side, toward the brain and the dura mater.

Yellow and blue are perceived the longest; but even these colors will become imperceptible. When the color-blindness has reached that point, everything appears *sepia*. Mr. Charcot has fully described the anatomy of that lesion.

Hemiopy, as found in the discases of the optic fascicles, and the result of the demi-decussation of the optic nerves at the level of the chiasma.

The fibres which do not cross at the chiasma, cross between the corpus-geniculatum and the corpora-quadrigemina; and thus each hemisphere receives all the fibres of vision from the opposite side. These fibres pass at first into the corpus-geniculatum, then afterward (*Gratiolet and Meyncrt*) pass below the optic thalamus and join, outward and backward, the common bundles of sensory fibres of the posterior portion of the internal capsule. The fibres of the olfactory (*Meynert*) bend backward with the bundles of the white anterior commissural fibres, pass below the cerebral ganglia, and definitely join the common bundle.

One thing is sure: that a lesion of the posteror portion of the internal capsule produces a complete hemianæsthesia of the opposite side of the diseased brain. Central hemianæsthesia differs from mesencephalic, by the existence of trouble in the sense of sight and olfaction in the former, while it does not exist in the latter.

Cerebral Hemichorea and its lesion, Athetosis.

Hemichorea is a trembling similar to the chorea of children, which takes hold of the paralyzed side, five or six months after an attack of apoplexy. It sometimes appears previous to the attack. Post-hemiplegic hemichorea must be distinguished from prehemiplegic. This peculiar tremor was first described by Weir Mitchel, and studied by Charcot and Raymond.

It begins in the upper limbs, then affects the face and sometimes extends to the lower limbs. It is only manifested when the patient executes voluntary movements; thus, when he wants to carry a glass of water to the mouth, the arm is seized with very extensive and rhythmical movements, and the water is thrown out of the vessel. In walking, the whole body receives shocks, and it becomes impossible, or at least very difficult, to continue. When the arm is extended, or when the hand is thrust into the pocket, it becomes immovable. During rest there is often a constant instability of the limbs. Athetosis, as described by Hammond, is nothing else but a peculiar post-hemiplegic chorea. It is characterized by incessant motion of the fingers and thumbs, however much the patient tries to steady them. Dr. Raymond has indicated the place of this lesion, and its cause, as being either a hæmorrhagic focus, or a softening, situated behind the thalamus opticus in the remotest part of the internal capsule. This locality being close to that where cerebral hemianæsthesia is caused, explains the frequent occurence of both symptoms at the same time.

Rendu and Gombault have given the following description of pre-hemiplegic hemichorea: The patient is repeatedly attacked with weakness and dizziness, which will cause him to fall down. After some of these attacks, his walk will become hesitating and unsteady, whilst the upper limbs will tremble. Such conditions may last several days; after this, motor hemiplegia will follow, either suddenly or slowly.

Differential Phenomena of Cortical and Cerebral Hemiplegia.

The character of cortical hemiplegia is, besides those enumerated above, 1. That it affects certain muscular groups only. The occulo-motor muscles are often perfectly unharmed. Aphasia is often wanting. 2. That there is never a permanent loss of sensation in the sensorium. 3. After a few days, there is a tendency to recover, and often complete restoration of health takes place.

Aphasia.

Physiologists have demonstrated that the third frontal convolution was the most important centrum of language. Ferrier's researches have shown that when *that* convolution of a monkey, or the corresponding one of lower animals, was excited by electricity, their lips and tongue were set in motion. Charcot has reported a case of a patient who was affected with simple aphasia, without any other paralysis, and in whose brain, after death, Broca discovered a circumscribed lesion of the convolution above named. This was caused by an embolus in the inferior external frontal artery, which supplied it.

The study of paralysis and convulsions, having their origin in cortical lesions (according to Charcot, Pitre, Grassiot, etc.), goes to prove that the cortical motor centra for the two upper limbs of the opposite side are situated in the *paraccntral lobule*, and *in the superior two-thirds of the ascending parietal* and *frontal convolutions*. The center of the movements of the lower portions of the face are situated in the inferior third of these same convolutions, in the neighborhood of the fissure of Sylvius.

HAEMATOMA DURAE MATRIS.

CEREBRAL TUBERCULOSIS.

Sec. I. *Tab. VI.*

Fig. 1.

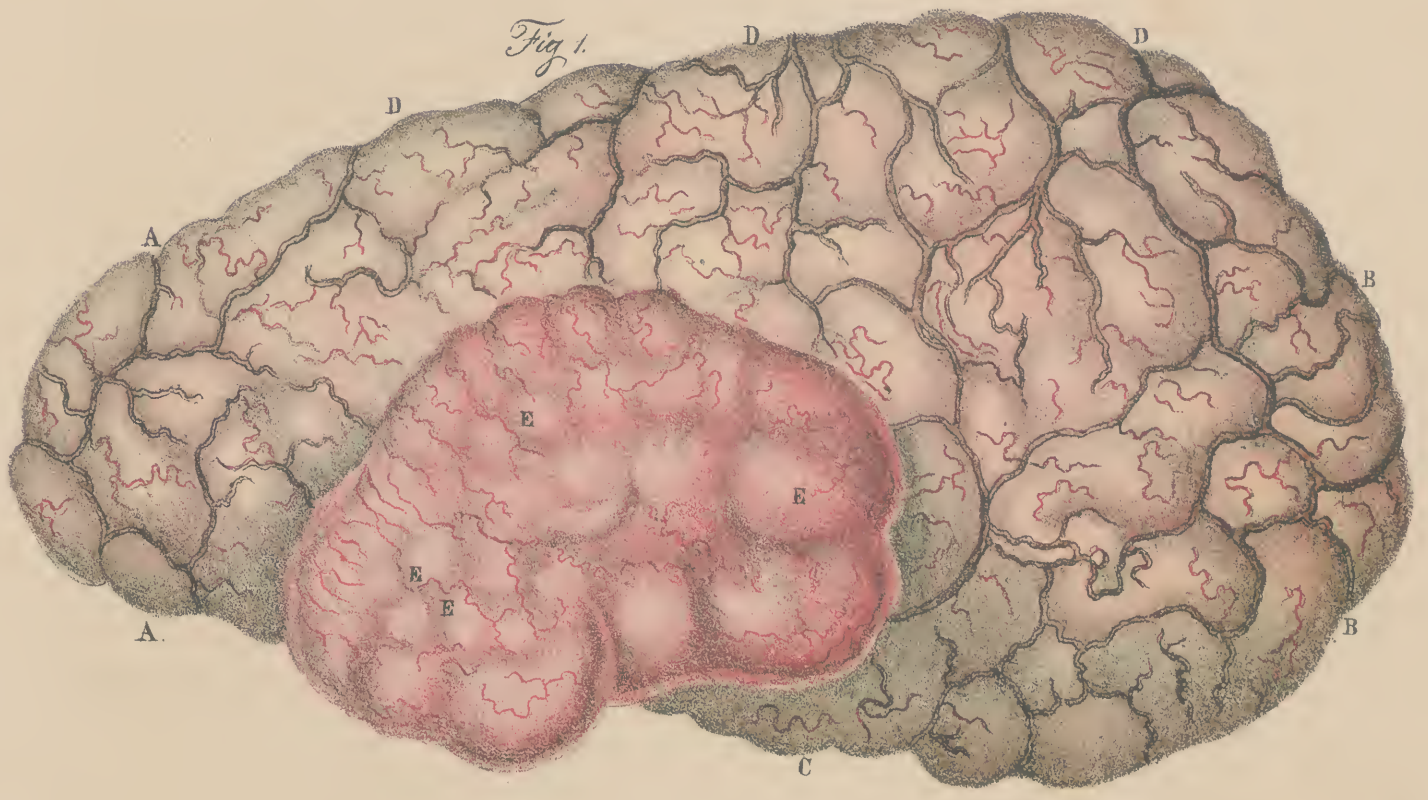


Fig. 2

Fig. 3.

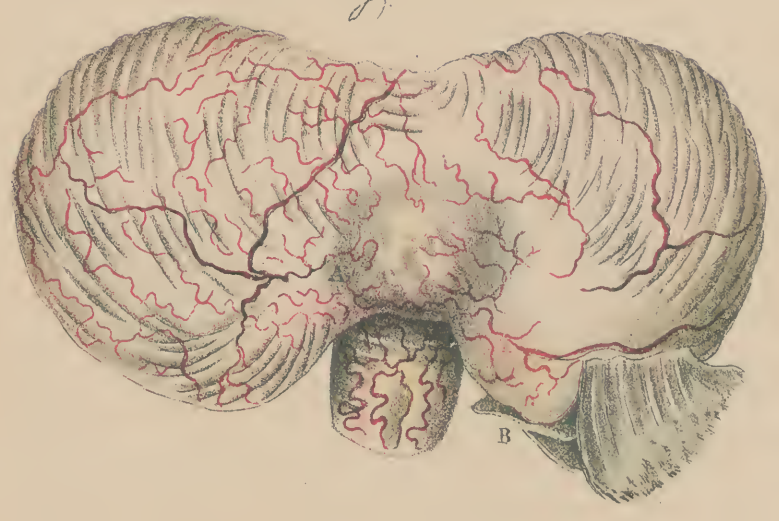
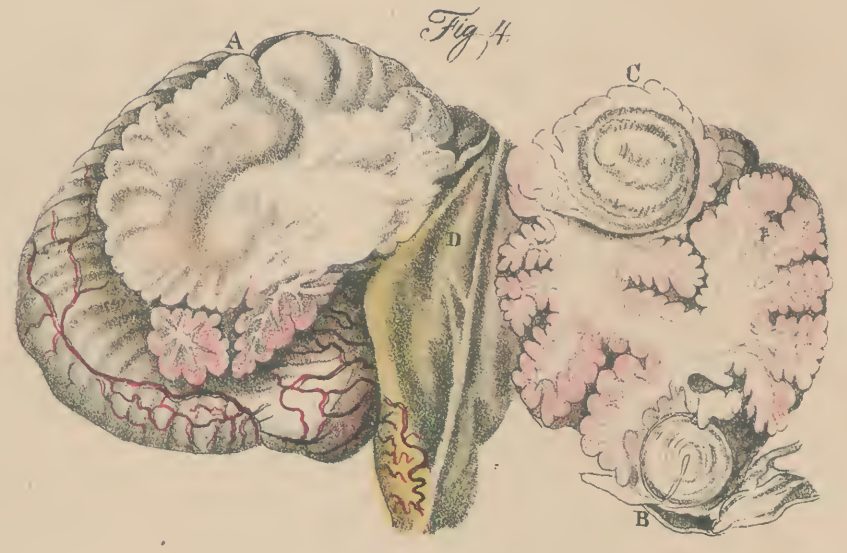


Fig. 4.



CONGENITAL HYDROCEPHALUS.

Apoplectic Focus in the Lateral Cerebral Ventricle.

HAEMORRHAGE IN DIGITAL CAVITY.

Sec. I. *Tab, VII.*

Fig. 1.

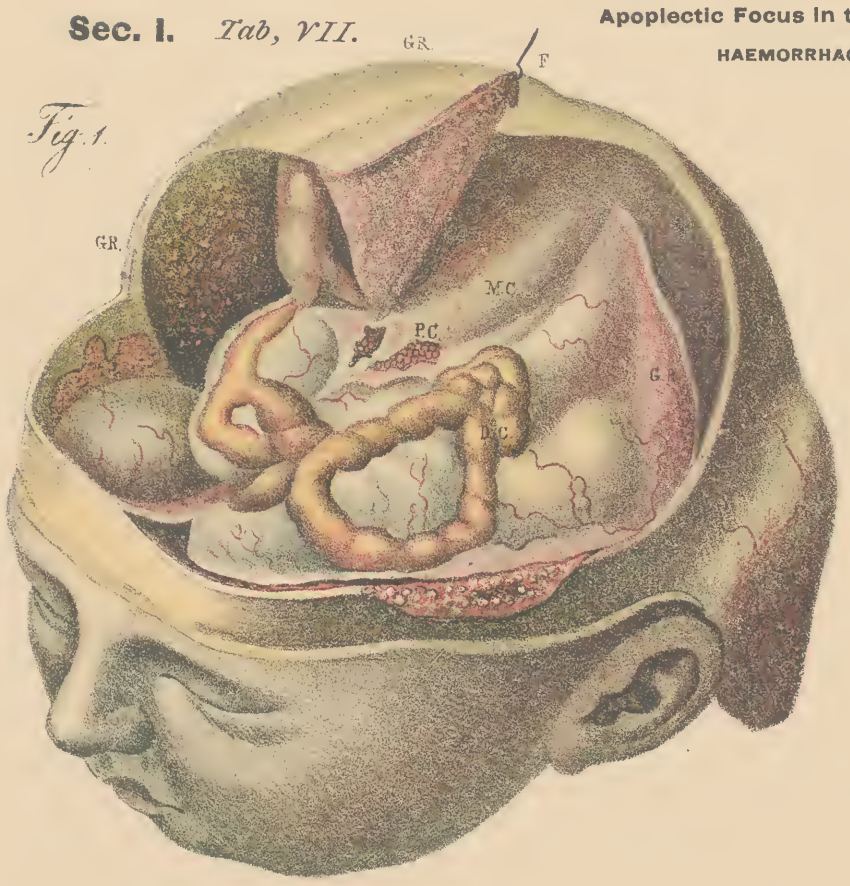


Fig. 1'.

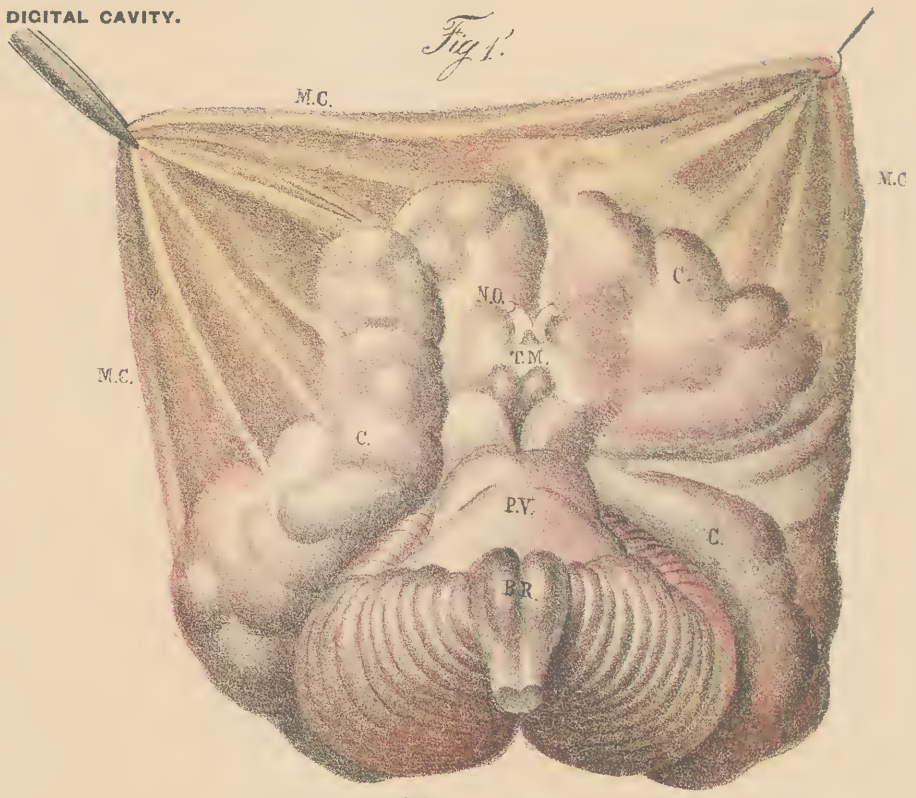


Fig 2.

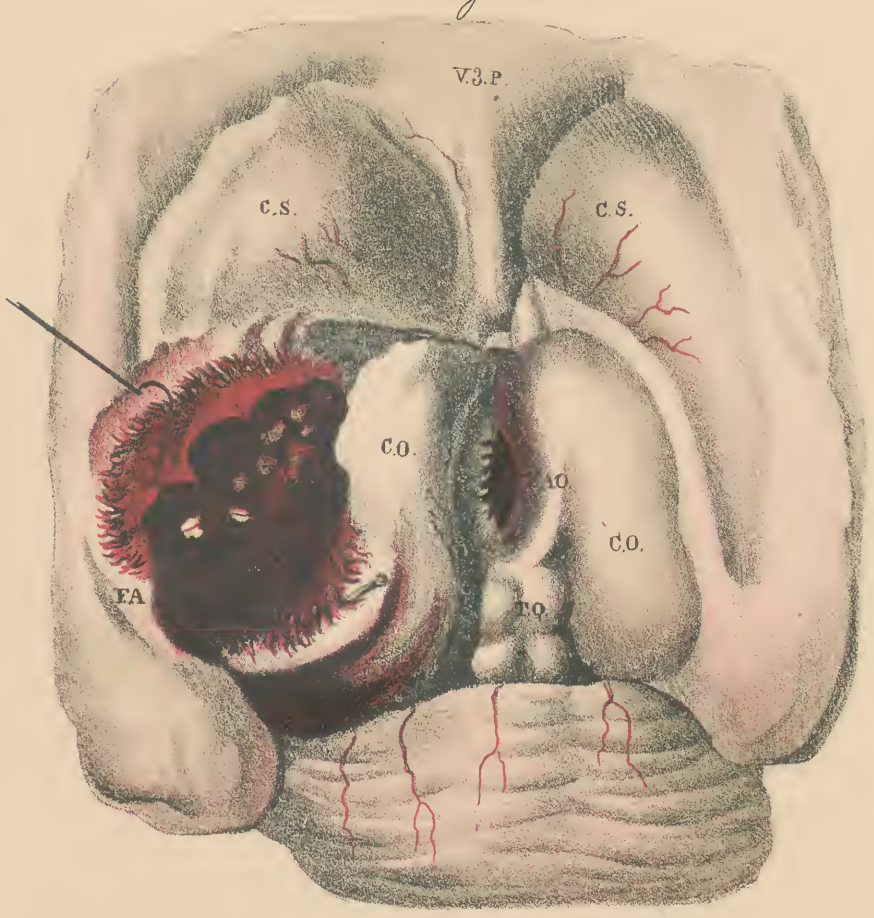


Fig 3.

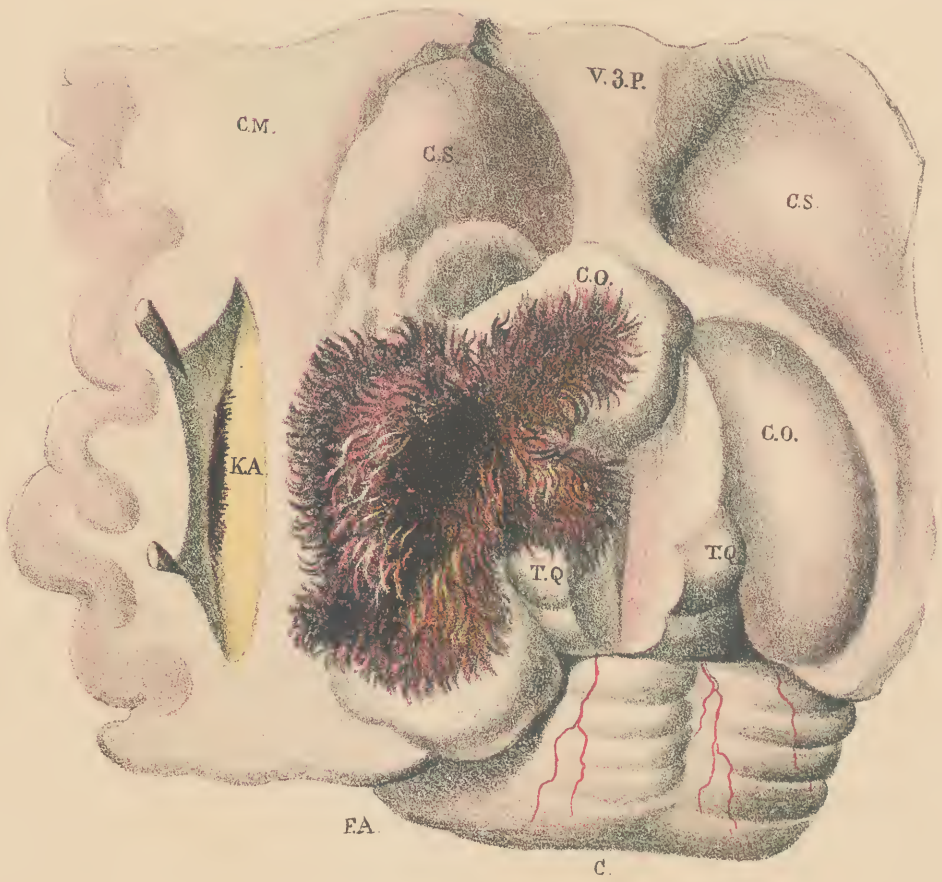


Fig.4.

Fig. 5

TABLE VII.

FIGS. 1, *1.—Hydrocephalus and Microcephalus.*

The cranium is smaller than usual, and ossification far advanced. The cavity contained a great quantity of serum. The cerebral hemispheres were obliterated and in their places existed a fine membrane covered with miliary granulations. (*M. C. G. R.*) Only two thin masses twisted together, cylindrical in form, and bent into irregular circles, occupied the middle cerebral fossæ. (*D. C.*) Microscopically examined, these masess proved to be cerebral substance, but in a high state of induration.

Below these masses, remnants of the thalamus opticus and corpus striatum were found on each side. The choroid plexus (*P. C.*) on each side, was also compressed, but still readily recognizable. The falx cerebri (*F.*) was also covered with the same granulations as the membrane described above.

Fig. 1 shows the basal surface of the brain. The cerebellum (*C. V. L.*) is intact. The *pyramids* of the medulla oblongata are absent, whilst the *olives* are thoroughly developed (*B. R.* medulla). The pons varoli (*P. V.*) and the cerebral and cerebellar peduncles are normal. The mamillary tubercles (*M. T.*) are of natural size.

The optic nerves were atrophied, whilst the olfactory were wanting altogether. The other nerves were normal. Some basal convolutions (*C. C.*) were in a good state of preservation. The membrane (*M. C.*) enclosing the remains of the cerebrum had neither the structure nor the form of the dura or pia mater.

FIGS. 2 AND 3.—*Ingravescent Apoplexy* (*Broadbent*).

CASE.—A powerfully-built man dropped, suddenly, insensible from his chair.

Symptoms.—Complete sensory and motor hemiplegia of the right side. Consciousness returned the day after the attack. *Deviation conjuguee* to the left. Lies motionless in bed, and answers all questions by signs. Face has a stupid expression. Involuntary discharge of urine. Pulse and respiration slow, but otherwise normal. Suffers no pain.

Next day. Same as before.

Third and fourth day. Same.

Fifth day, the eyes open, but fixed. Respiration and pulse a little more frequent, but feeble. Excessive perspiration followed by very easy death.

Post-mortem Autopsy.—Very extensive apoplectic focus (*F. A.* Fig. 2.) in the depth of the left thalamus opticus. (*C. O.*) The lateral ventricle contained bloody serum. By raising up the fornix (*V.* 3 p.) and removing the choroid membrane, a rent was found in the middle cerebral ventricle (*F. A. O.*), which contained a large clot.

The right and left corpora striata (*C. S. C. S.*), the right thalamus (*C. O.*), and the corpora quadragemina (*T. Q.*), and the remainder of the brain perfectly normal.

(The morbid phenomena were due to hæmorrhage taking place between the lenticular nucleus and the external capsule. *Broadbent.*)

Fig. 2 shows the apoplectic focus widely laid open and its walls exposed, the remnant of the optic thalamus is turned up and the right tubercula quadragemina (*C. O. T. Q.*) separated from the left.

When slightly magnified, the focus shows many tufts of capillaries and other smaller vessels of the thalamus opticus. Besides this apoplexy a cyst existed in (*Ka.*) the hemisphere.

FIGS. 4 AND 5.—*Hemianæsthesia and left incomplete hemiplegia.*

CASE.—A woman of 72 years. Before her entering the Maison Royale de Sante (Paris, France) she is said to have suffered from an attack of apoplexy with complete left-sided hemiplegia.

Symptoms since.—Hemianæsthesia, hemiplegia incomplete. The limbs are more stiff than lame. Lies in bed, but feels perfectly comfortable. Is perfectly rational; in her sleep she is slightly delirious.

Bedsores; followed by gangrene; she dies twenty-five days after the attack.

Post-mortem.—The lower right limb was enormously infiltrated with pus, consequent upon a severe erysipelas shortly before death. A number of metastatic abscesses in both lower extremities. A large fibrous tumor in the uterus. Pelvic vessels very much dilated; some atheromatous.

In the cranial cavity were found the following changes:

In the right parietal fossa the convex surface of the brain had a brown color, very soft and fluctuating. When it was incised a quantity of a brownish serum ran out. The clotted blood contained in the same cavity was of the same color. The apoplectic process, occupied the whole medullary center of the right hemisphere, and extended in the direction of the convolutions, some of which it destroyed; and was only checked by the *Genu* of the *corpus callosum.*

In the lateral ventricle it communicated by a narrow fissure with the *digital cavity*, which it had enormously distended, and which contained a quantity of clotted blood. A cyst was found in the cellular tissue (*M. C. V.*), of a bright yellow color. A large quantity of vascular tufts could be seen by slightly magnifying that part of the cavity. A number of small arteries, the walls of which were partly ossified (*A. O.*), existed in the same cavity. In the center of the thalamus opticus a soft fibrous tumor (*C. A.*) also existed.

PATHOLOGICAL ANATOMY OF THE CEREBRO-SPINAL MEMBRANES.

In the later stages of the disease (pachymeningitis) the neomembrane will be found to consist of several layers. The innermost will be of a character just described. The next following, outside of it, is distinguished by a greater abundance of cellular elements in the parenchyma, and by narrower vessels. The third or outermost layer is composed of tough, shining, connective tissue fibers, nearly as closely knit as that of the dura mater itself; in fact it partakes of the same general character as neoplastic formations found in inflamed connective, or serous tissue (f. ex. the pericardium).

There is a peculiarity in pachymeningitis: that it is inclined to relapse; and has a tendency to form upon the cicatrized structure new pseudo-plastic deposit as upon the *dura* itself. This explains why persons affected with this disease so very seldom perfectly recover. It seems that when the blood vessels have become contracted by cicatricial tissue, a new exudation is produced, by hyperæmia, caused by increased blood pressure in the cranial cavity.

The peculiarity of the newly-formed membrane makes repeated hæmorrhages a histological necessity.

In very thin pseudo-membranes, hæmorrhagic spots penetrate a great portion of its mass. In thicker and thickest, hæmorrhagic centra are even more frequent, and of far greater extent. They are in some places several inches in diameter and fully as deep.

At one time they were thought to be *hæmatomata*, and designated as such (Tab. VI, Fig. 1). They are lodged interstitially in the meshes of the neomembranes. The larger and the largest ones exist in the dilated spaces between the layers or upon the dura mater itself. A fine gossamer tissue encloses them like a sac.

Occasionally blood coagula are found between the arachnoidal surfaces by infiltration from the internal dural surface.

Such large extravasates necessarily prove fatal.

It very seldom happens that the whole pseudo-membrane is converted into pus. Where the hæmorrhages are not too severe, the membrane undergoes retrogressive metamorphosis and leaves behind a great number of spots full of pigment (Tab.V, Figs. 2, 5). Such changed neoplasms are often met by the side of newly-forming, and differ from each other by their color. The vessels in old ones are very often perfectly obliterated (Tab.V, Fig. 2),

TABLE VIII.

FIGS. 1, 2 AND 3. — *Softening of the Brain. Inflammation of the Superior Longitudinal Sinus and of the Veins emptying into it. Hæmorrhage of the Gray and White Cerebral Substances.*

CASE.—A young woman of 20 years was taken sick on April 20, 1855.

Symptoms. — Affected with enteric typhoid; watery stools. Pulse, 128. Respiration, 18. Temperature, 102°, Fahr. Tongue red at the edges, very dry.

May 2d. Symptoms of pleuro-pneumonia on right side.

May 3d. Is very morose and very indifferent to her surroundings. Complains of pain in the right shoulder. The head is often turned to the right or to the left. Has severe pain on the slightest movements of the head.

May 9th. Decubitus on the left side. The trunk is bent forward, the thighs flexed upon the abdomen. All voluntary muscles in a state of tonic contraction. Is perfectly stiff in the back. A disease of the spine is supposed to exist.

May 10th. Continued complaint of pain. Sometimes inarticulate sounds. The eyes fixed upward and to the right. Pupils extremely dilated. Convulsive and epileptiform movements of the left side, especially the upper limbs. The left thumb is strongly pressed into the closed palm during the spasms, which succeed each other at almost regular intervals. The right side is not convulsed. Is insensible to pain, or to pricking with a needle, all over the whole body. Continues to moan and utter inarticulate groans.

May 11th. Comatose; but still moans. Respiration very frequent; likewise the pulse. The head regains its normal position, the eyes are closed, but the pupils continue dilated. The left side absolutely stiff. No convulsion.

May 12th. Still comatose. Tries to put out the tongue, but is unable to do so. The pupils less dilated than the day previous. The left arm half contracted. By trying to approach it to the trunk it is seized with rhythmic convulsions.

May 13th. Gradually, general paralysis takes place. Is utterly insensible. Respiration extremely frequent. Pulse small and quick. Dies in the night.

Post-mortem.—In the abdominal viscera, three circular ulcers in the small intestine, about ten inches above the ilio-coecal valve. Mesenteric glands swollen and very red, but not softened. In the chest, red hepatization of the inferior lobe of the right lung.

In the head, the superior longitudinal sinus (Fig. 1, *S. L.*) is filled with clotted blood, adhering to the vascular walls. All the veins emptying into it are similarly filled, the lateral sinus likewise. After the removal of the meningiæ, several apoplectic focuses are found to have spread over several convolutions of the left cerebral hemisphere, adjoining the longitudinal sinus (*A. C. A. C.*). They passed from the gray into the white substance. A portion of the right hemisphere was softened; and, here and there, hæmorrhagic points were scattered, sometimes in groups, sometimes singly, in both hemispheres. The spinal marrow was not examined. (N. B. Had this been examined, a severe lesion would certainly have been discovered therein, as the stiffness of the neck and dilated pupils well indicated.)

FIG. 2. — *Softening of the Brain.*

CASE. — M, 75 years old, is suddenly attacked by paralysis of the tongue; the mouth is twisted to the left. The right arm is perfectly stiff. No trouble in the left side, nor in the lower extremities. No loss of sensibility. Very small, slow pulse. Temperature of the body below the normal. Intellect not at all disturbed; yet he is unable to answer questions. (*Aphasia.*)

The next day patient suddenly loses consciousness. Stertorous and irregular breathing. The right arm perfectly stiff. Involuntary movements of the left arm. Is sensitive to pain. Pulse very irregular, weak; sometimes very frequent, sometimes very slow.

In the night, alternate contraction and relaxation of the limbs. The eyes roll in their sockets. Purple color of face. Dies on the third day after the attack.

Post-mortem Autopsy.—Several convolutions of the left hemisphere are affected with red softening of the gray substance. The softened portion is adherent to the membranes and can be readily removed with them. The white substance beneath it seems unaltered, whilst at other points is much changed, and extends to the center of the same lobe. In the right hemisphere one convolution, adjacent to the great median fissure, showed also a loss of substance similar to the one described above. It seems as if a contracted cicatrice had taken the place once occupied by a portion of the inflamed brain. It seemed to have nothing to do with the present attack.

Fig. 3 represents the right cerebral hemisphere of an old woman who died in a hospital. She was affected with left-sided hemiplegia for many years. Immediate cause of her death was senile gangrene of the whole left hip. She was rational to the last moments of her life.

The figure represents a section in an antero-posterior direction. It shows plainly the great loss of cerebral substance, and its replacement by yellowish connective tissue, containing some blood vessels. This cicatricial tissue was of feeble cohesion and broke down very readily. A quantity of yellow serum ran out of it after it burst. The cerebral tissue surrounding this cicatrice was perfectly normal and the heteroplastic part was perfectly circumscribed. A close examination showed that the cicatricial tissue formed in the line of the convolutions, which must have been gradually obliterated and as gradually replaced by foreign substance.

C. H. D. Right cerebral hemisphere. *C.* Cerebellum.
F. N. Neoformation. *S. L.* Longitudinal sinus.
H. G. Left hemisphere. *A. C.* Apoplectic center.
R. Shrunk convolutions. *M.* Middle lobe.

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but their former course is indicated by the pigment left behind.

Serous effusions in the membranes happen very seldom; and when they do exist they partake of the nature of interstitial hydrops.

Leptomeningitis.

Although the pia mater forms the general covering and vascular storehouse of the brain, yet its several sections present, clinically, such individual character in inflammatory conditions, that their special pathology must be separately described. In fact, the membrane is but very rarely affected in its totality.

We find inflammation of the pia mater over the convex surface of the brain (*external hydrocephalus*); at the base and of the fissure of Sylvius, (*basilar meningitis*), the ependyma, the ventricles; of the choroid plexus with ventricular œdema (*internal hydrocephalus*).

(Tables I and II of this section present some examples of the latter.)

For the better understanding of the peculiarities of the different lesions of the brain, and its membranes, it is necessary to study the conditions of circulation in the cranial cavity.

As that cavity is limited, with little exception, on all sides by the hard tissue of the skull, and a definite amount of its space is to be occupied by its contents, it is plain that if any liquid were to enter and occupy a portion of that space, it would displace some of the liquid or solid contents of the cavity. As the blood is carried into the brain by the carotids and vertebral arteries and from it by the sinuses and jugulars, a constantly equable blood pressure might be expected to exist in the cranial cavity, and, theoretically, no hyperæmia nor exudation, to any extent, could take place in the brain and its membranes. Indeed, some have denied the existence of hyperæmia in the cranial cavity, in the face of all observed facts proving the contrary.

The arterial walls of the brain are exceedingly thin, whilst the sinuses are very thick walled.

RED SOFTENING OF THE BRAIN.

HETEROPLASIA OF CEREBRAL CONVOLUTIONS.

Fig. 1.

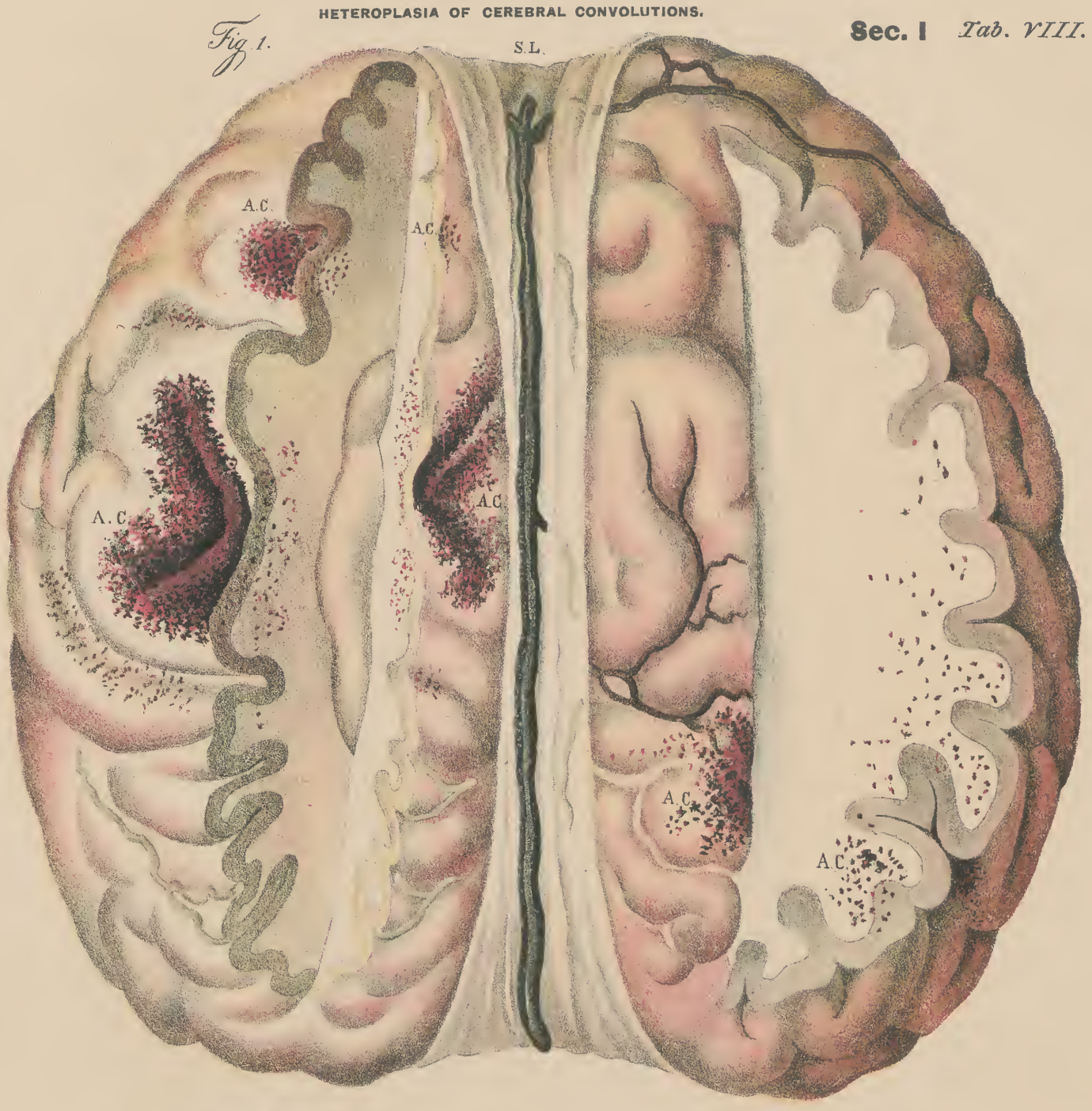
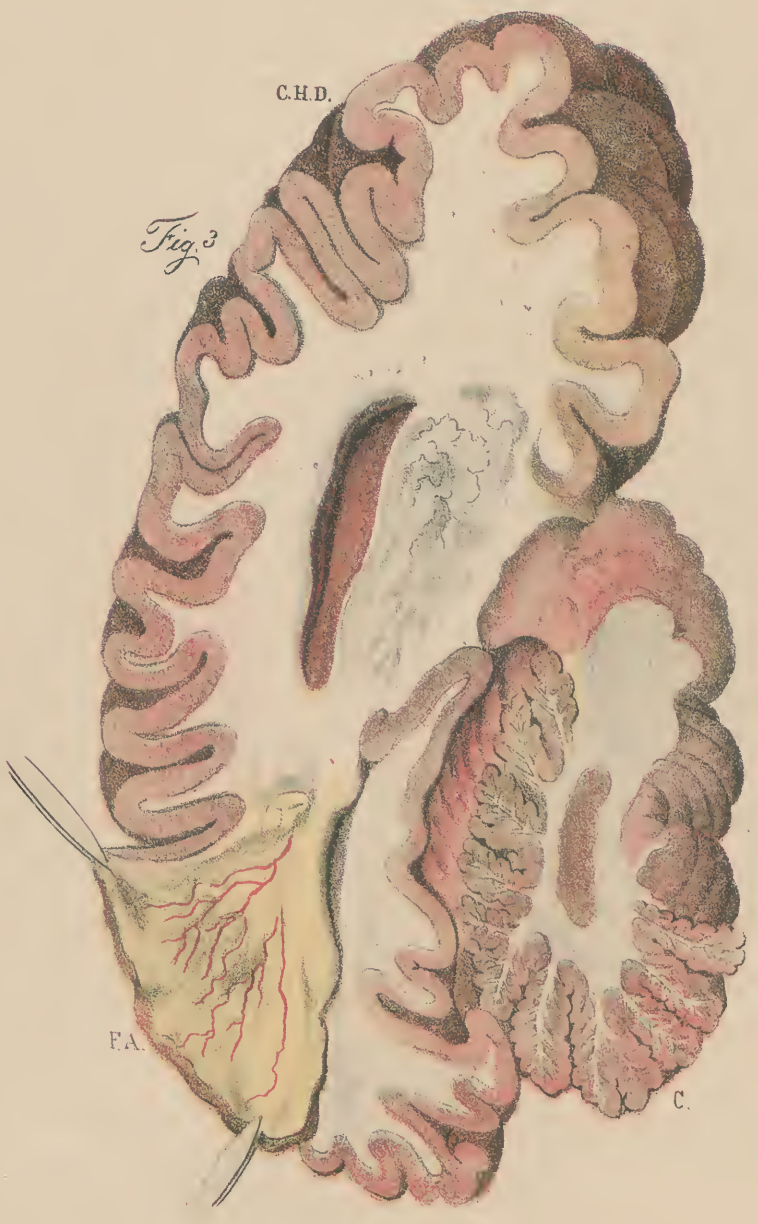


Fig. 2.

Fig. 3.



Sec. I. *Tab. IX.*

COMPLETE HEMIPLEGIA AND APHASIA.

INJURIES OF PONS VAROLI AND PYRAMIDS.

Fig. 1

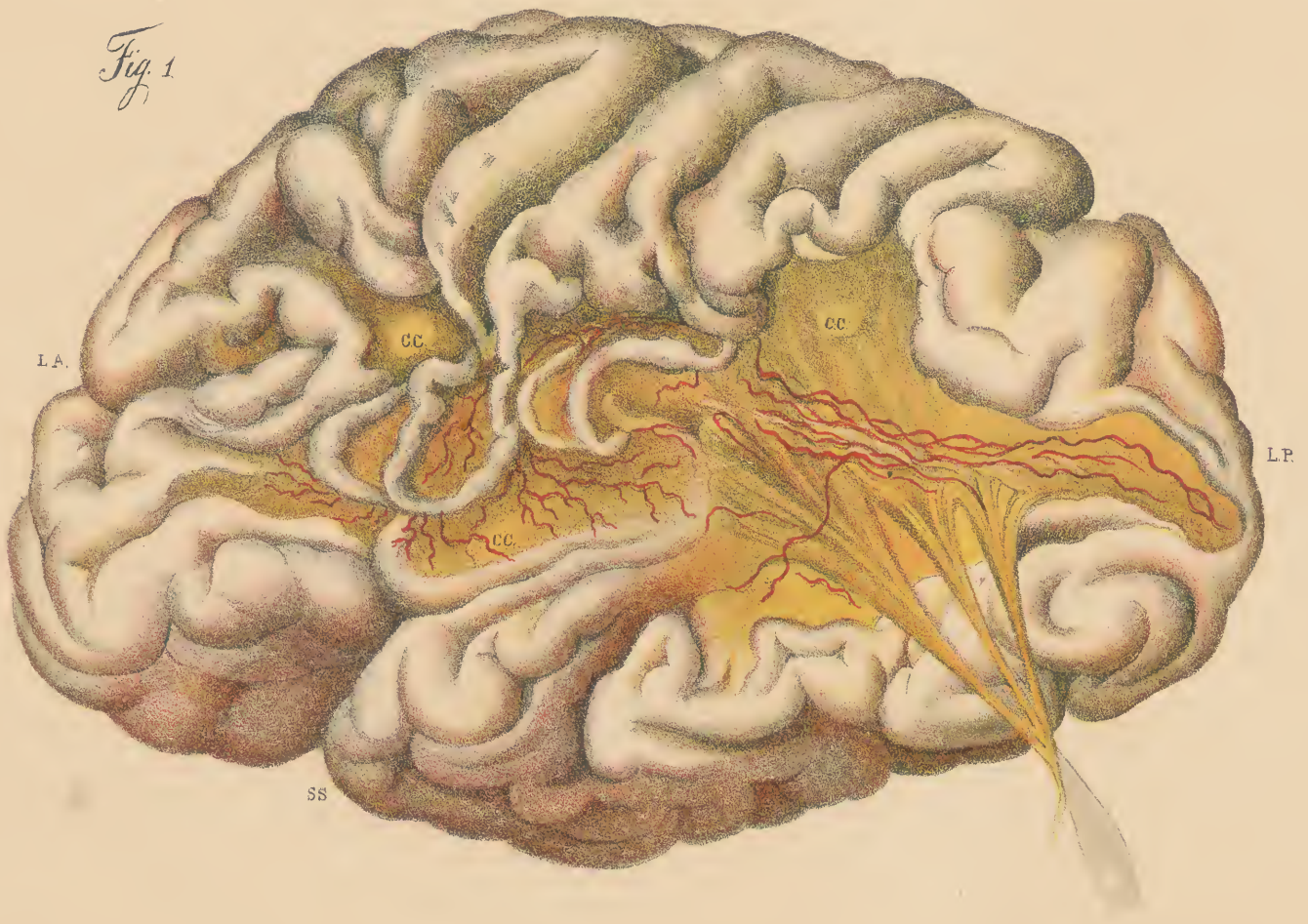


Fig. 2.

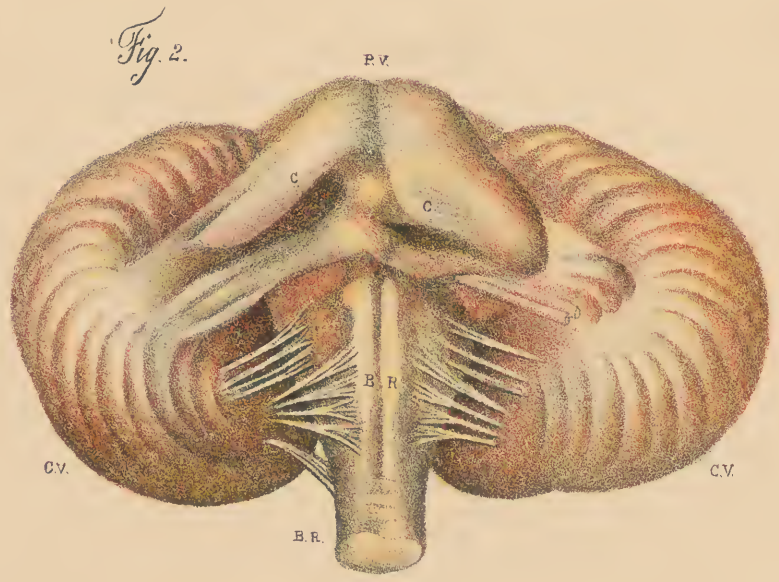


Fig. 3

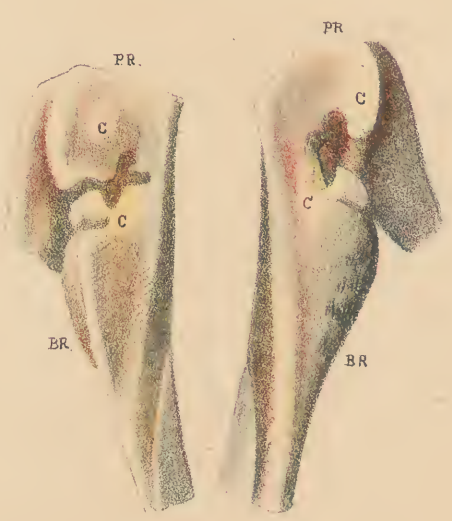


Fig. 4 P.A.C.

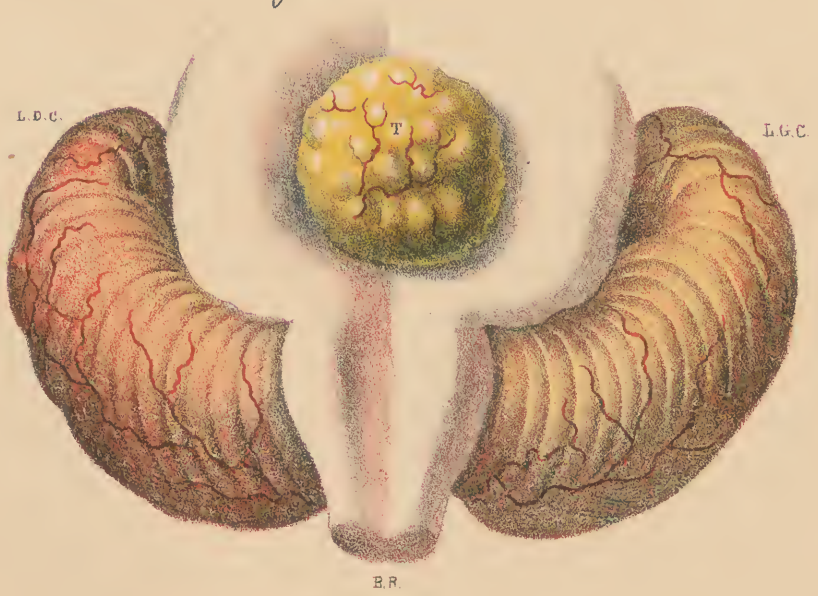


Fig 5

W.M. DONALDSON & CO. LITH. CHILD

Table IX.

Fig. 1.—*Complete Right-sided Hemiplegia and Aphasia.*

Case.—A woman about 55 years old.

Symptoms.—Total right-sided hemiplegia; left side normal. Is unable to articulate a single syllable. Intellect and sensation unimpaired. Is always in good humor. Has often attacks of epilepsy. A month before her death she became melancholy, drowsy, and lost her appetite. She complained of no pain. Two days previous to her death she was seized with very violent epileptiform convulsions, which destroyed her life.

Post-mortem Autopsy.—Brain, external surface of left cerebral hemisphere (the side opposite to the paralyzed), shows (Fig. 1.) destruction of parts of frontal, parietal, spheno-temporal and occipital lobes; and of portions of the island of reil (lobule of corpus striatum). A yellowish (chamois skin color) membrane occupying the place of the destroyed cerebral matter. The membrane was soft and of different thickness in the different localities. A great quantity of vessels of variable caliber traversed it, in several directions. The brain substance proper, immediately beneath it, seemed injected with the same yellowish coloring matter as the membrane.

The convolutions injured were:

1. Inferior frontal. 2. First sphenoidal. 3. Third sphenoidal. 4. Posterior parietal. 5. Second sphenoidal. 6. Second occipital. There were also destroyed the corpus striatum on the same side, two-thirds of the optic thalamus, and a portion of the ventricular membrane. The anterior and posterior corpora geniculata were normal. The optic nerves likewise (hence vision was unimpaired). The neoplastic membrane occupied the place of the obliterated corpus striatum; passed outward, lined the fissure of Sylvius, thence to the external surface and covered the white substance in place of the cortex.

There was some destruction in the right hemisphere, but not nearly so extensive.

A considerable portion of the brain was sclerotized.

Fig. 1 represents the external surface of the left cerebral hemisphere. *L. A.* Anterior lobe. *L. P.* Posterior lobe. *C. C.* Destroyed convolutions replaced by neomembrane. *S. S.* Fissure of Sylvius.

Pathology of the Case.

The destruction of the inferior frontal or the convolution of *Broca*, which is the seat of articulate language, well accounts for the aphasia of the patient (Ferrier). The complete hemiplegia of the right side had its locality of lesion in the motor centrum, constituting the internal capsule, from the middle and anterior peduncular fibres, in their passage between the lenticular and caudate nucleus (Carville and Duret). It is seen that the destruction or injury has reached the external surface of the lenticular nucleus and the *island of Reil*, and has destroyed the claustrum; lesions of this locality, producing complete hemiplegia of the side opposite to the injury.

That the sensation was not impaired was due to the fact that the greatest portion of the optic thalamus was uninjured, and likely, where the posterior peduncular fibres expand on a plane with the thalamus, no lesion existed, for here *the central origin of hemianæsthesia is located* (Duret and Veysiere). The epileptiform spasms indicated a spread of the disease to the *pons*, an impediment to the circulation of the cortex, and pressure upon some convolutions, which constitute motor centra of the extremities.

Figs. 2 and 3.—*Injuries of the Pons and Pyramids.*

Case.—Mary D., a corpulent and seemingly vigorous woman of 52, affected with complete right-sided hemiplegia. Paralyzed limbs perfectly stiff. Incomplete hemianæsthesia of left side. Total assymbolic aphasia. The patient, by the greatest efforts, produces but unintelligible sounds. Sheds abundant tears on the slightest provocation, sobs violently, and seems in a terrible state of mental agony. Her face is strongly flushed, and she suffers frequently from convulsions of the respiratory muscles. Disphagia and pharyngeal spasms occur very often.

Involuntary alvine discharges.

Although able to move her left limbs, they do not seem completely under her control. Her intellect is not at all disturbed. Her appetite and sleep are very good. This condition was observed by Dr. Regnier, an Interne at the Salpetriere, for about a year.

She often complained, by signs, of headache, and was relieved by slight phlebotomy (?) Six weeks prior to her death she commenced to lose her appetite and became feverish. A large ulcer had formed upon the sacrum, which occasioned heavy fever. Hypostatic pneumonia, with very harassing cough, also developed. The ulcer speedily mortified and spread over the whole gluteal region, invading the surface and the deeper parts. Severe dyspnœa and constant cough with inability to expectorate. Died from asphyxia.

Autopsy shortly after death.—The skull enormously thick. Small reddish points (hæmorrhagic foci) on inner surface of dura mater. Veins very tortuous. Much serum in subarachnoidal spaces of cranial and spinal cavities, filling the sulci and fissures.

An old pseudo-membrane was adherent to the inner dural surface.

The cerebral surface seemed normal (?) The gray matter of some of its convolutions seemed softened (post lethae).

The pons varoli (Fig. 2.) was small and deformed, presenting two cicatrices and loss of substance on the posterior inferior border. The right cerebellar peduncle and adjacent portion of the pons was partly softened and partly atrophied. The upper pontine surface was atrophied one-half. The left peduncle slightly altered. On the left, the pyramids perfectly obliterated; on the right, much atrophied. The olivary bodies very voluminous and sclerotic. The hypoglossal nerve of a grayish color. The medulla oblongata was separated from the pons by a deep fissure (Fig. 3). The loss of substance on the right side of the pons not nearly so great as on the left. A grayish-yellow pseudo-membrane occupied the place of the obliterated cerebral substance.

A small-sized tumor existed on the right lateral surface of the falx cerebri. The spinal marrow normal.

The heart of normal size. On the semilunar valve-flaps of the aorta many excrescencies and calcareous scales. A true aneurism of the thoracic aorta (the sac about three inches long), filled with lamellated clots. The calcareous scales were also spread over the *intima* of the aorta.

P. V. Pons varoli. *C. V.* Cerebellum. *C. R.* Crura cerebri. *B. R.* Medulla oblongata. *C.* Crura cerebelli.

The brain and the spinal marrow do not nearly fill the whole of the cranial and spinal cavities. The foramen magnum is only half occupied by the spinal marrow. The whole central nervous structure is encased by the pia mater. The free surface of the pia is invested by the arachnoid membrane, which lies in almost immediate contact with the inner surface of the dura mater. Only a minimal quantity of serum normally exists between the two latter surfaces.

Between the dura and the cerebro-spinal central substance the pia mater occupies all the space. Within the so-called subarachnoidal space, there circulates the cerebro-spinal liquid and freely communicates with the cerebral ventricles by the foramen Magendii and the lateral orfices of the fourth ventricle. (*Key and Retzius.*)

This liquid passes from the ventricular spaces, through the Pachionic granules, into the intervenous spaces of the dura mater and empties from there through some lymphatic canals of the head into those of the nasal mucous membrane. (*Naunyn and Schreiber, Arch. f. exp. Path., vol.* 1.)

FIGS. 4 AND 5.

CASE.—J. M., 11 years old. Strong child. Is suddenly seized with a violent fever, lasting several weeks. During convalescence the lower limbs became œdomatous, and abdominal dropsy developed. Was tapped four times. Shortly after, he began to complain of violent lancinating pain in the back part of the head, and became suddenly affected with strabismus. Amblyopia, gradually followed by complete loss of sight. Articulation of sound became very difficult; deviation of the mouth to the left, and partial right-sided hemiplegia supervened.

In about six weeks strabismus and inability to articulate disappeared; but dizziness and great numbness, with partial loss of consciousness followed. Vision remained very imperfect, and deviation of the mouth became far more marked. The right limbs became perfectly paralyzed, œdomatous and cold.

About ten months later, on admission to the hospital, the following symptoms were recorded: Occipital cephalalgia, deviation of the mouth to the left, incomplete right-sided paralysis.

He is able to move his fingers, but can neither lift his arm or keep it raised when lifted by the waiter. Incomplete right-sided hemianæsthesia. Gives correct answers to questions, but articulates very slowly and laboriously. Left side normal. Amblyopia on both eyes. The right pupil is dilated, the left contracted. General health bad; thoroughly developed cachexia. A month later he dies in a collapse.

Post-mortem.—Cerebrum and its membranes normal. A viscid effusion in the subarachnoidal space.

In the pons there was a spherical tumor of about an inch in each diameter. It had a concentric structure, and resembled certain forms of urinary calculus. It occupied mostly the left half of the pons and extended into the crus cerebri of the same side. The right crus was also invaded by a similar, though smaller, tumor; equally so the anterior wall of the fourth ventricle.

An incision into the pons and the medulla showed another tumor to have lifted the valve of Vieussens, and compressed from below upward, the corpora quadrigemina.

Chronic adhesive pericarditis and several large-sized tubercles were found. Numerous ulcers of the smaller intestine, and chronic adhesive peritonitis; enlarged and indurated mesenteric glands also existed.

TABLE X.

FIG. 1.—*Cerebellar Tumor.*

CASE.—A blacksmith, 50 years old, suffered a number of years from chronic bronchitis. The whole integument slightly cyanotic. Otherwise nothing abnormal about it. Nothing abnormal in function of motion or sensation, except that his gait is unsteady and shuffling. Sight good, nothing abnormal in fundus *oculi*. No strabismus. Sense of hearing, right side, slightly defective. He is suddenly seized with excessive debility, becomes insensible and dies.

Post-mortem.—Besides the anticipated lung and heart trouble, in the thorax, the cranial cavity showed partial synechia of the dura and pia mater; much serum in the sub-arachnoidal space and ventricles; a lobulated tumor of solid consistence existed on the right anterior inferior border of the cerebellum, of about three inches long, about two and a half wide, and about half an inch in thickness. It extended over the anterior and inferior surface of the pons and upon the pedunculi cerebri, partly compressed them, also the cranial nerves, with the exception of the first, second, and ninth pair. It extended also into the internal auditory meatus.

FIG. 2.—*Atrophy and Cicatrices of Cerebellar Lobes.*

CASE.—A woman who had suffered from right-sided hemiplegia, was also affected with chronic catarrhal pneumonia, in consequence of which she died.

Post-mortem.—The inferior surface of the left lobe presented a considerable loss of the cortical substance (*L. G.*). A brownish membrane partly replaced the destroyed substance (*C. C.*). The medullary portion did not seem to be injured. The inferior vermiform process not disturbed.

Pathology.—The symptoms during life did not indicate anything peculiar to cerebellar lesions. Besides the hemiplegia (which very rarely is due to strictly cerebellar lesion), no disturbance of cerebral function noticeable.

Fig. 3. Section of cerebellum. *P. A.* Pons varoli. *A. B.* Basilar artery. *B. R.* Medulla. *L. G.* Left lobe. *L. D.* Right lobe.

Fig. 4 presents the superior surface of the cerebellum of a child about 10 years old. He suffered for a long time from occipital cephalalgia. Other symptoms rather obscure. Died of general debility and milliary pulmonic tuberculosis.

Two tuberculous masses (*T. O. T. O.*) were lodged in the depth of the organ. Although pressing upon the cerebral substance they produced barely any histological change upon it (?) The color of the tumor was of a greenish-yellow. The interior of the masses of the tumor had a lamellated structure.

FIG. 5.

CASE.—A seventy-year-old man.

The posterior and inferior surfaces of the cerebellum were destroyed. Atrophy injured the adjacent portions. This extended in front to the pons, and laterally to the medulla oblongata. Symptoms: Was affected with incomplete right-sided hemiplegia. *Permanent deviation conjuguee* of the eyes to the right.

Post-mortem Autopsy.—Showed the above named lesion and more or less degeneracy of the external coat of basilar and vertebral arteries. A great many calcareous scales covered the perivascular sheaths of those vessels and the vessels themselves.

Fig. 6, section of Fig. 1, showing the direction of the nerve fascicles passing in different directions through the pons. (*C.* Cerebellum. *C. P. V.* Crura and pons varoli.)

This liquid (the cerebro-spinal) plays a very important role, when morbidly increased pressure takes place in the sub-arachnoidal and ventricular spaces. For, as it surrounds all the large and small vessels, passing through the sub-arachnoidal spaces and stands in open communication with the ventricles, it will constitute a uniform pressure all over those spaces, and local pressure, produced by tumors, extravasates, etc., will be rendered harmless (*Huguenin*, *Althans*, *Bergman*).

When acute or chronic exudates produce pressure upon the cerebral membranes, liquid is always found in the sub-arachnoidal spaces and in the ventricles. Likewise is the effusion of the meningiæ and hydrocephalus found in those spaces, and not in the sub-dural cavity. When any liquid is ever found in the latter — and that happens very rarely — it is due to transudation from the sub-arachnoidal into the sub-dural space.

The highly important and exceedingly numerous labors of Leyden, Jolly, Pagenstecher, Duret and Cramer, have demonstrated that no general cerebral pressure has ever been produced, by increasing it, in the sub-arachnoidal and ventricular spaces, but always by an increase in the sub-dural cavity; for from this space it cannot easily diffuse itself *all over the* brain, and thus relieve the local compression.

As stated before, the foramen magnum is not altogether occupied by the spinal marrow; the cerebral liquid can, therefore, readily communicate with the spinal cavity. When one cavity is filled, the liquid passes into the other, and *vice versa*. This gives room for oscillating blood pressure, and thus hyperæmia and exudation can only exist in circumscribed localities, and only to a certain degree. When both cavities are filled, no more pressure from exudation can take place, but solid neoplasmata may form or extravasation take place, at the expense of the intercranial organs, and in this manner fatal anæmia by pressure may be brought about.

CEREBELLAR TUMOR. ATROPHY OF CEREBELLAR LOBES.

Sec. I. *Tab. X.*

Fig. 1.

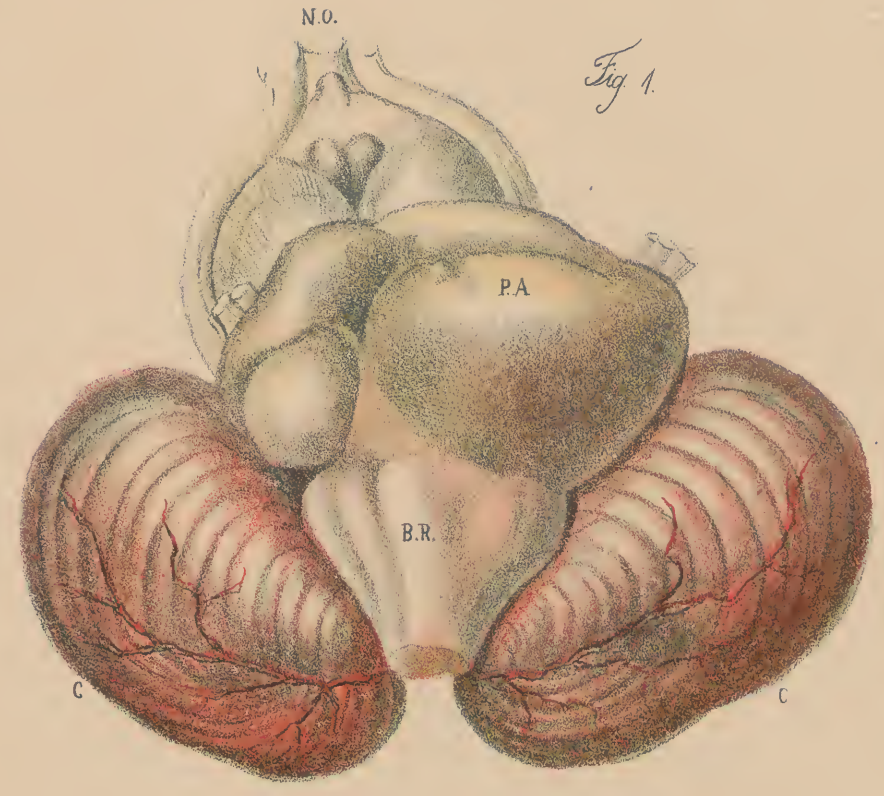


Fig. 6.

Fig. 2.

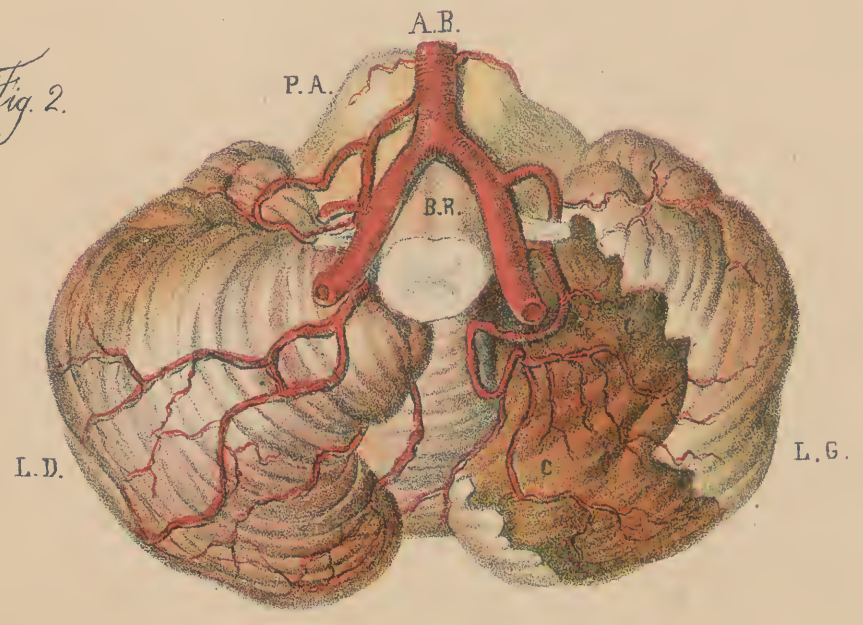


Fig. 3.

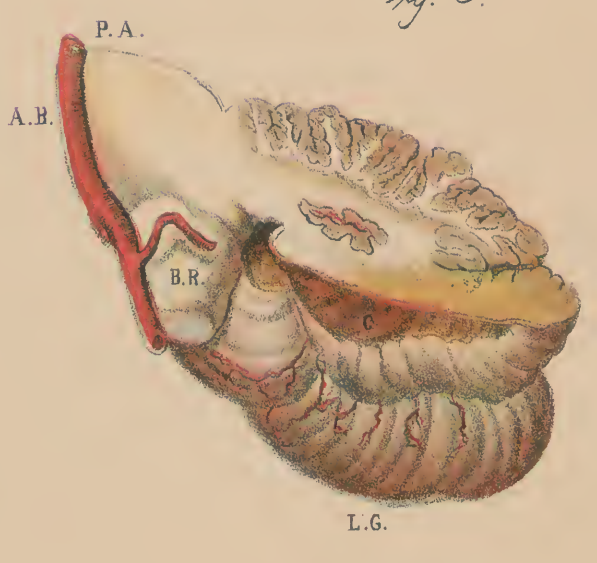


Fig. 4.

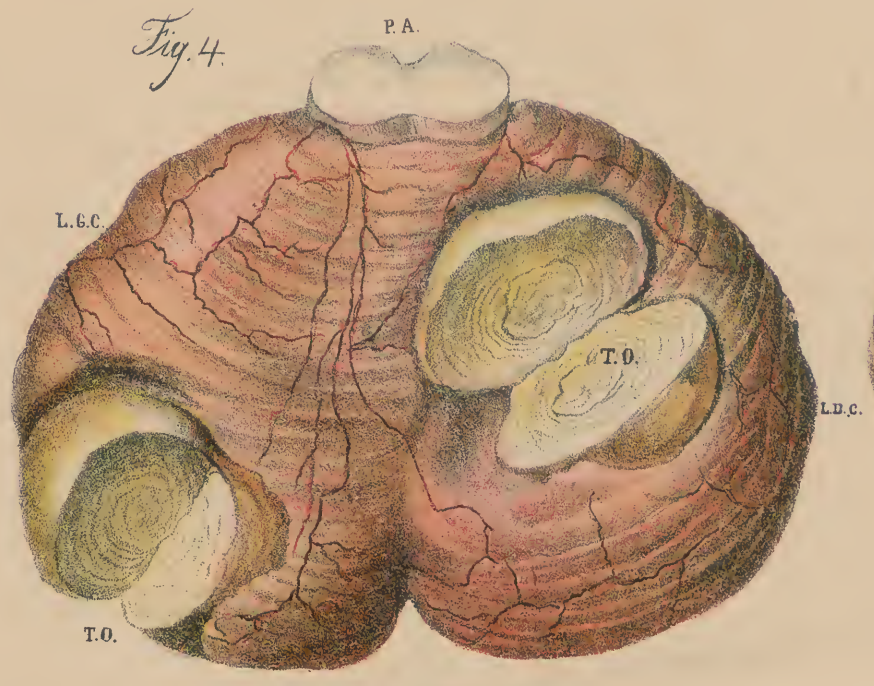
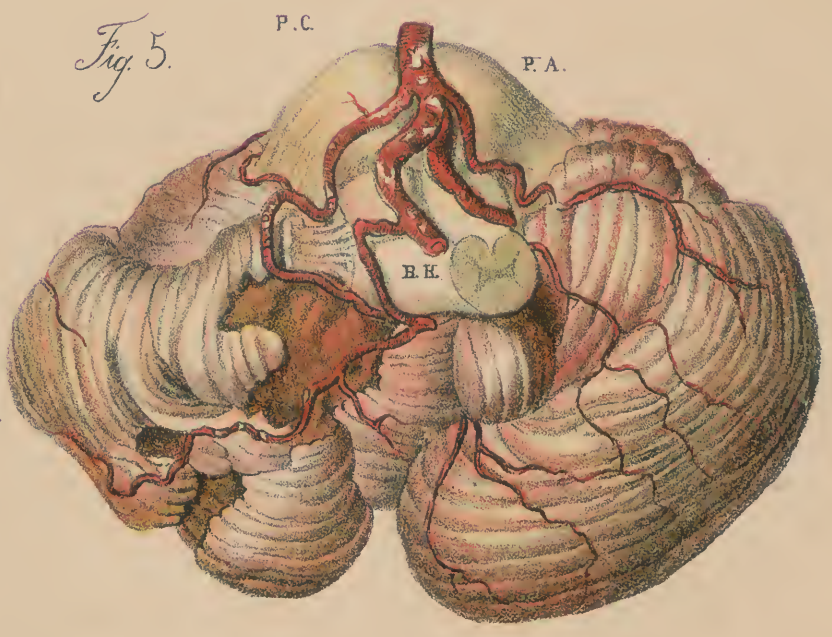


Fig. 5.



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COMPRESSION OF CEREBELLUM AND PONS VAROLI.

Sec. I. *Tab. XI.*

Fig. 1.

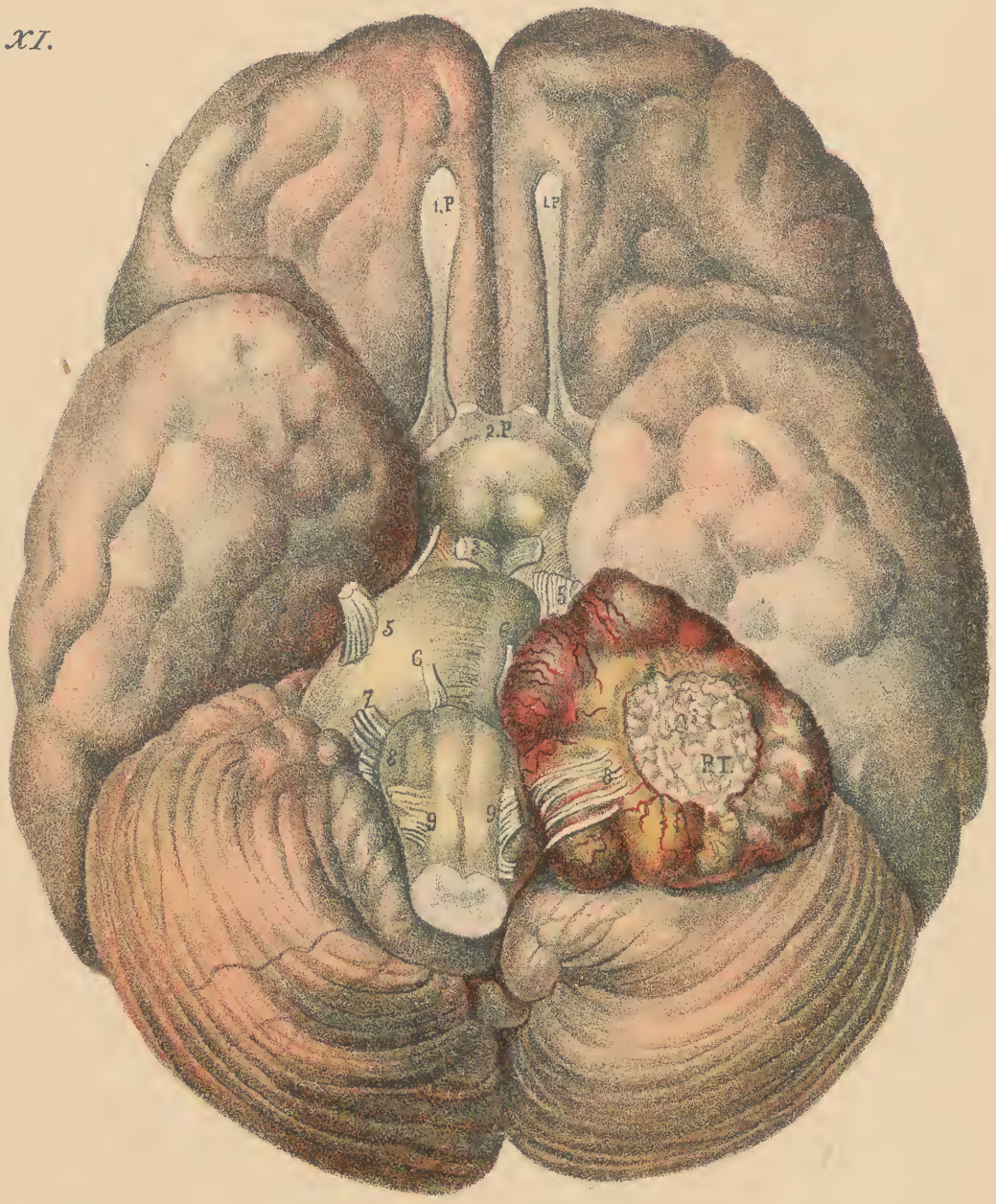

Fig. 4

Fig. 3

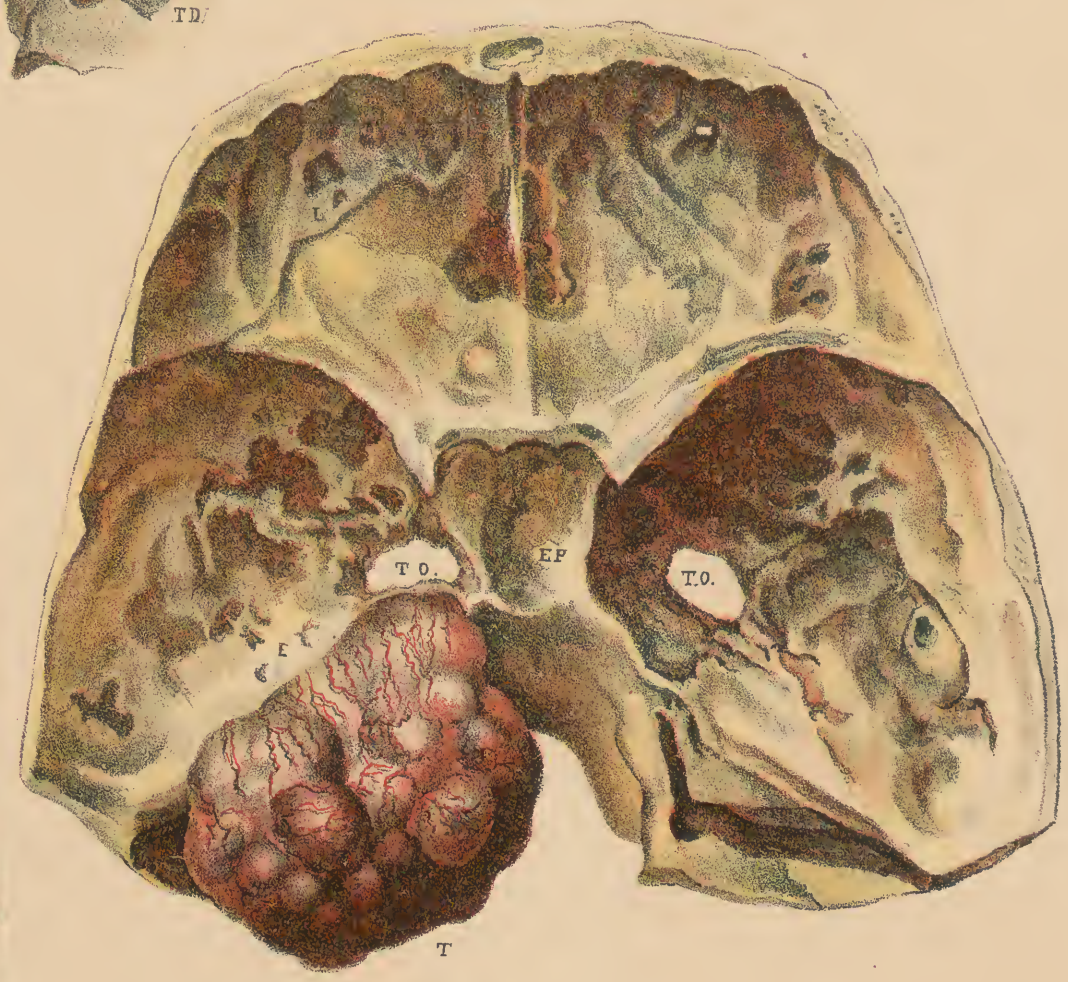

Fig. 2.

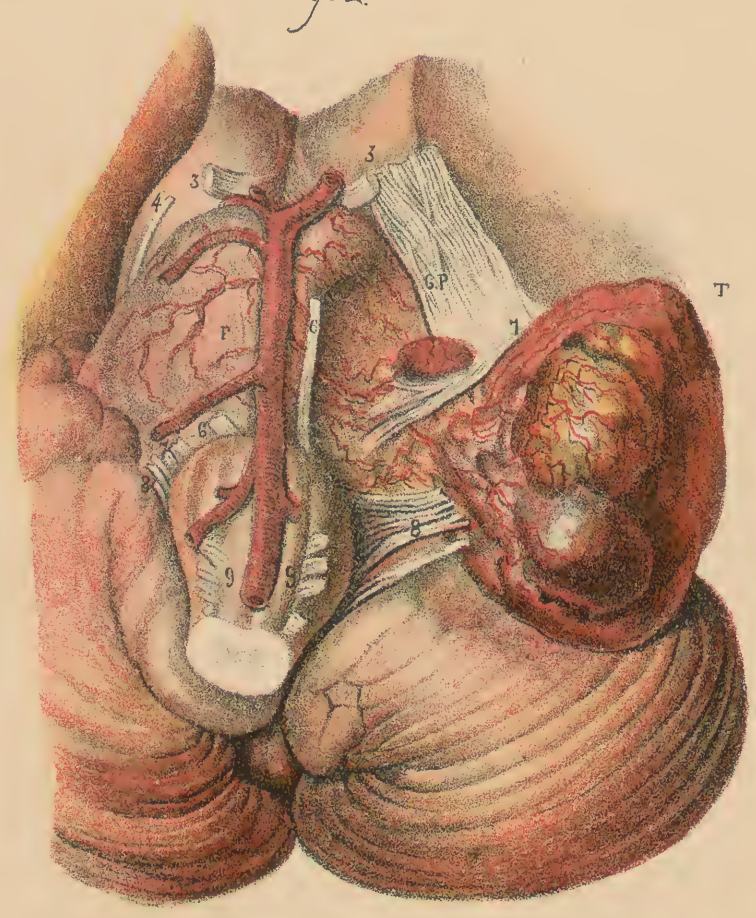

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Table XI.

Compression of a portion of the Cerebellum and Pons Varoli by a Tumor.

Case.—Amable A. A woman of 26 years.

History.—At the age of 19 she became affected with repeated attacks of violent headache, which would last about a week and be relieved by cold application to the head. She became slightly deaf in the left ear.

At the age of 20 years, amblyopia in the right eye developed; a year later convulsive contraction of the left cheek, with deviation of the mouth to the right. Gradual loss of vision and increase of intensity of the spasms of the cheek. General health good up to ten months before her death. At that time formication in the right cheek, more aggravated headache and considerable pain in the left thigh. The contraction extended to the upper limb. Shortly before death she presented the following symptoms:

Complete amaurosis. Loss of sense of smell and hearing. Complains of violent pain in the top of the head, numbness in the face, alternating with pain in the left thigh. Intellect undisturbed. No loss of common sensation. From time to time facial spasms, alternating with stiffness of the upper limbs, especially the left.

In a few weeks she became very feeble, and had to stay in bed all the time. Towards the last, gradual paralysis of the tongue, and delirium. Her appetite, which was good before, now began to fail; more or less constant vomiting, and finally, perfect loss of appetite. Occasionally she had lucid moments and her speech was unimpeded. A few days before her death she seemed surprisingly improved, but was suddenly seized with convulsions, became unconscious, and died without a struggle.

Post-mortem.—The cranial cavity:

In the left occipital fossa a hard, fibrous tumor, of the dura mater, situated beneath the tentorium cerebelli, was found to have compressed, from below upward, the lateral left half of the pons, the pedunculi cerebelli and cerebri on the same side and the medulla oblongata.

Fig. 1 shows the tumor imbedded in the encephalic mass. The pneumogastric, spinal accessory and glossopharyngeus were displaced by it. Their substance did not seem morbidly altered. The seventh and fifth were compressed, displaced and flattened. The sixth pair were displaced but not altered. The left olfactory nerve (1st *P.*) was partly atrophied. The right still more so. The optic nerves (2nd *P.*) were completely atrophied and their mass of a gray color (sclerotic).

The seventh and eighth pairs were detached from their place of emergence and compressed. The cerebellar peduncles and the cerebellar lamellae were both compressed and atrophied. The smaller root of the fifth nerve was perfectly obliterated, and the larger very much out of place; both altered and of a gray color. This gray color extended into the inferior maxillary division, after its emergence from Gasser's ganglion. The tumor, of which the pedicle (*P. T.* Fig. 1.) offered some traces of ossification, and which was buried in the cavity represented in (Fig. 4*a*.), could be readily separated from its bed: the portion of the dura mater projecting into the internal auditory meatus. This cavity (*T. E.* Fig. 4.) had effaced the auditory meatus and opened communication between this, the foramen lacerum posterior and the carotid canal. The auditory and facial nerves after their entrance into the petrous bone were perfectly destroyed.

Fig. 3 represents the inner surface of the skull. It shows that portions of the different fossæ were greatly modified, and brought about changes in the cortex of the brain and portions of the dura mater. There were the following alterations of the inner surface: The basilar fossa was depressed, portions of the sphenoid destroyed. The sella turcica (*F. P.*) was much enlarged and contained the pituitary body with a depressed portion of the middle ventricle; and was filled with serum. Portions of the sphenoidal surface were very brittle, and some already broken. The orbital and spheno-temporal fossæ were deeply corroded and perforated. The two foramina ovala (*I. O.*) considerably widened. The foramen magnum was eccentrically widened on one side. The ethmoidal fossæ were dug out deeper and wider, and filled with very numerous holes. These had exceedingly weakened the basis of the skull and made it very brittle. The orbital fossæ were perforated from one to the other.

The frontal sinuses were nearly all gone. The optic foramina narrowed. The substance of the brain was lodged in all the depressions produced by corrosion of the bone.

1st *P.* First pair. 2nd *P.* Second pair. 3rd *P.* Third pair. 4th *P.* Fourth pair. 5th *P.* Fifth pair. 6th *P.* Sixth pair. 7th *P.* Seventh pair. 8th *P.* Eighth pair. 9th *P.* Ninth pair. *P. T.* Pedicle of tumor. *T. E.* Ethmoidal fossa. *T. O.* Optic foramen. *T.* Tumor. *T. D.* Foramen lacerum posterior. *C. A. I.* Internal auditory meatus.

What is ordinarily called inflammation of the brain is really only *an acute hyperæmia and purulent infiltration* of the pia mater (see Tab. I. Figs. 3, 4, 5, of this section and Tab. V. Figs. 1, 2, 3, 4, 5, of same).

(These are well pronounced cases of purulent leptomeningitis; there is hyperæmia of all the vessels of the pia mater and infiltration of pus into the lymphatic spaces.) The pus is strictly confined to the parenchyma of the membrane and does not surpass its limits, the perivascular spaces are filled with it, and along each vein a string of pus can be seen. These strings eventually form networks, and these constitute membranes which fill out the sulci and press asunder the gyri, and form perfect casts of the cerebral surfaces.

Basiliar Meningitis.

Differing from the above, both in form and nature, is basilar meningitis. It is nearly always associated with tuberculosis. (Tab. I. Fig. 7. Sec. I.) It has therefore been called *tubercular meningitis*. Its chief character is the development of tubercles in the outer coat of the vessels (Tab. I. Fig. 8. Sec. I.) of the pia mater, especially that portion of the membrane which invests the sulci and median fissure of the base of the brain. The tubercles are most numerous in the vascular sheaths in the Fissure of Sylvius (Tab. I. Fig. 6. Sec. I.). The tubercular masses not only protrude from the outer surfaces of the vessels, but they also drive the middle and inner coat into the vascular cavity, often perforate these coats and occupy the perforated spaces. The consequence of it is gradual obstruction of the vascular cavities and weakening of its walls. This alone would suffice to produce enormous disturbance in the cerebral circulation. But let an acute hyperæmia take place, and increased blood pressure in the dilated vessels, then a hæmorrhage will be caused by the weakened portions and an acute inflammatory condition of the still normally preserved parts of the vessels. It will be found that *the tuberculous condition of the vessels leads to basilar meningitis of the pia mater.*

As a rule the cortex cerebri partakes in tubercular meningitis (Tab. I. Fig. 5. Sec. I.). This is due, in part, to the vessels of the pia mater passing into the cortex without any perivascular structure of any thickness, and partly by the continuity of the inflammatory infiltrate.

The tubercular degeneration of the cerebral vessels proper, often passes to the border of the center of *Vieussens*, and there produces enormons dilatation and often severe hæmorrhage, leading to destruction and degeneration of the cortex itself.

Rokitansky and Rindfleish mention cases where tuberculous vessels caused enormous hæmorrhages, and red softening of the cortex around the fissure of *Sylvius.*

Venous Hyperæmia and Œdema of the Pia Mater (External Hydrocephalus).

The veins of the pia mater may undergo permanent dilatation and relaxation by long-standing irritative hyperæmia. (For instance, in chronic alcoholism, psychoses, etc.) The results of such conditions are similiar to those produced by mechanical impediments to the flow of the venous blood to the heart (Tab. IV. Figs. 1, 2. Sec. I.): thrombosis of the longitudinal sinus and of the other large sinuses. The seat of the hyperæmia will be found to be in the region of the veins. As the longitudinal sinus is not capable of much dilatation or change of caliber, the branches emptying into it will be found so much more dilated and ectatic, and are extented in manner to form tortuous windings. The smaller branchlets

PLATE XII.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

CASE.—M. Darge, a female, forty years old.

History.—Has been two years in the Salpetriere, Paris. First attack of the disease six years prior to her death. Early symptoms: Monoplegia of the left thigh, soon followed by monoplegia of right thigh. Subsequently, attack of paresis of both upper extremities. Tremor in each limb. Is able to use them to some extent. No paralysis of sensation in either extremity. Intellect undisturbed. Is occasionally seized with hystero-epilepsy. Intermittent paresis of the tongue, with some impediment of speech, and inability of deglutition. No cephalalgia. Hearing, good. Amblyopia on both eyes. A few weeks before death she had an attack of pleuro-pneumonia. Gangrene developed on bedsore of sacrum. Death from asphyxia.

Autopsy.—Thoracic cavity. Tuberculous lungs. Acute lobular pneumonia. Œdema pulmonum.

Cerebro-spinal cavity: Fig. 1. Anterior aspect of spinal marrow, medulla oblongata and pons Varoli. Anterior pyramids sclerotic, gray color. Right olivary body (*O. D.*) sclerotic, except at one point where there is white softening. Left olive (*O. G.*), sound. Restiform bodies, sclerotic. (A section of the medulla oblongata showed the gray sclerotic alteration to have extended to the whole pyramids, the right olivary body and the restiform cords.) The nuclei of the hypo-glossal and glosso-pharyngeal nerves were in the same condition; and at their origin the nerve fibers were devoid of axis cylinders. The gray alteration (*T. G.*) extended in the spinal marrow, to a distance of two inches below the medulla oblongata, on its anterior aspect. The columns had cord-like appearances. From the origin of the first dorsal to the third lumbar nerves, the spinal marrow was softened, but contained perfectly sound and indurated portions. The softened parts had a semi-transparent, jelly-like appearance.

Fig. 2 represents the lower portion of the spinal marrow, anteriorly, which, also, underwent gray induration. The middle portion nearly normal.

Fig. 3 the posterior aspect of spinal marrow, etc. The posterior spinal roots nearly normal (no loss of sensation). The posterior surface of the medulla oblongata, sclerotic.

FIG. 4.—*Progressive Bulbar Paralysis.*

CASE.—A man of sixty-two years, affected with chronic rheumatism; suddenly seized with dizziness and headache.

Symptoms.—Is unable to swallow anything; attempt at deglutition produces very violent cough and hiccup. Both pupils normal. Lower portion of face much relaxed. Tremor of lower jaw. Stammering, and very indistinct speech. Is able to stick out his tongue slowly, without trembling. Soft palate very much relaxed and anæmic. Movements of arms free, but slow, and partly choreatic. No ataxia. Is unable to stand without support. Severe pain in the back of the head and neck. The symptoms gradually increase in intensity. There is involuntary discharge of stools and urine. Dies in six days after the attack.

Post-mortem.—No lesion perceptible to the naked eye in the cerebro-spinal cavity. Microscopic examination shows a lesion which begins in the middle portion of the olivary bodies, and extends to their upper borders. At the highest point of its extension it reached from the floor of the fourth ventricle to the pyramids on both sides of the raphe, rather more to the left than to the right; lower down it stopped at the fourth ventricle. Upward it did not pass the line of the pons. The morbid alteration consisted of granular degeneration, interspersed with round cells. At the edges of the disease-focus re-active demarcation, showing increase and tumification of the nerve fibers, took place. *Glia* cells, partly undergoing granular changes, intermixed with these. The vessels and ganglionic cells were not affected. At the upper edge of the altered focus, there is a place where the nerve fibres were completely obliterated and replaced by a vascular membrane. (*T. G.* Gray sclerosis.) (*T. R.* Red softening.)

FIGS. 5 and 6.—*Incipient Bulbar Paralysis from Syphilis.*

CASE.—A man of sixty-five years.

History.—He was affected with syphilis twenty-four years. Ten years ago had an attack of syphilitic rheumatism. An ulcer, situated in the sub-clavicular fossa, caused him much suffering. Six months ago he was seized with difficulty of swallowing, moderate disturbance of speech, and unsteadiness of gait. Suffered from loss of sleep, and severe affections of the glandular and osseous tissue. There was, also, arterial sclerosis. The spinous process of the sixth cervical vertebra seemed loose, and readily movable. A fluctuating tumor near the seventh cervical vertebra. Sudden attack of cerebral apoplexy—and death.

Post-mortem.—The apparently loose spinous process proved to be but a gummatous tumor. Skull very much thickened. The dura mater adherent to the skull. Gyri of cerebrum much flattened. Vessels on convex surface sclerotic. Extensive hæmorrhagic infiltration into the sub-arachnoidal space of the upper surface of the cerebellum. Hæmorrhage in the right lateral ventricle. The greater portion of the corpus striatum and thalamus opticus of that side obliterated. Hæmorrhage into the left lateral ventricle. Pons and medulla oblongata swollen and soft. The whole surface of the pons, to the apex of the fourth ventricle, as well as the upper surface of the processus cerebelli ad pontem, covered with multiple focal softening, from disseminated minute hæmorrhages. Microscopic examination, from the anterior border of the pons to the middle of the fourth ventricle, comprising the origin of the glosso-pharyngeal nerve, showed disseminated focal hæmorrhagic myelitis; the inflammatory process was most intensely developed in the region of the facial nerve. The arteries were sclerotic, and contained many small aneurismal dilatations. Numerous minute hæmorrhages in the peri-vascular lymphatic sheaths. The ganglionic cells, in the most intensely diseased portions, had undergone turbid swelling. Their nuclei had nearly disappeared, and contained, near the blood vessels, a large quantity of round cells. *4m. V.* Fourth ventricle. *C. P.* Crura cerebri.

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enter into all sorts of arrangements, they form tufts, networks, etc. (Tab. IV. Fig. 1, Sec. I.) The immediate results of such a condition is profuse serous effusion, or dropsy of the pia mater. This liquid is perfectly limpid, and contains a mere trace of albumen. It occupies the meshes of the membrane, and is very readily emptied by the slightest puncture of the tissue. Considering the smallness of the place it occupies, the quantity of the effusion is sometimes out of all proportion. It must necessarily occupy the space which the cranial viscera should occupy, displace those which do exist, or obliterate them.

The consequences of œdema will depend on the length of time it took to form and diffuse. When large quantities are slowly diffused into the sub-arachnoidal space, the brain gains time to accommodate itself to the compressed condition, and will afterward bear such pressure for an astonishingly long time. The gyri become parted from each other very widely, and the sulci become excavated and form broad troughs. Gradually the substance of the hemisphere diminishes from the ventricle toward the cortex; and loses sometimes over one-half of its mass. Its space is then occupied by the dropsical liquid.

But when the effusion takes place suddenly and in a short time, it is either speedily absorbed or the compression produced by it proves fatal to the patient. In chronic hydrocephalus the pia mater undergoes morbid changes, especially in the region of its vessels. It becomes opaque and assumes a milky appearance.

It becomes indurated both on its arachnoidal and cerebral surfaces, and its vessels entering the brain, as well as the cortex in the region of the vessels, become dry and tough like buckskin (*Cuir de Daim, of Cruveilhier*). The lateral ventricles are nearly always found dilated and filled with serum. The same is the case in tubercular meningitis. The source of the infiltrate is, no doubt, the choroid plexus. For very many of the vessels of the choroid are covered with tubercles, and the tuberculous degeneration of these vessels is just as liable to produce serous effusion and hæmorrhages in the ventricles as in the region of the fissure of Sylvius.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

Progressive Bulbar Paralysis.

INCIPIENT BULBAR PARALYSIS.

Fig. 5.

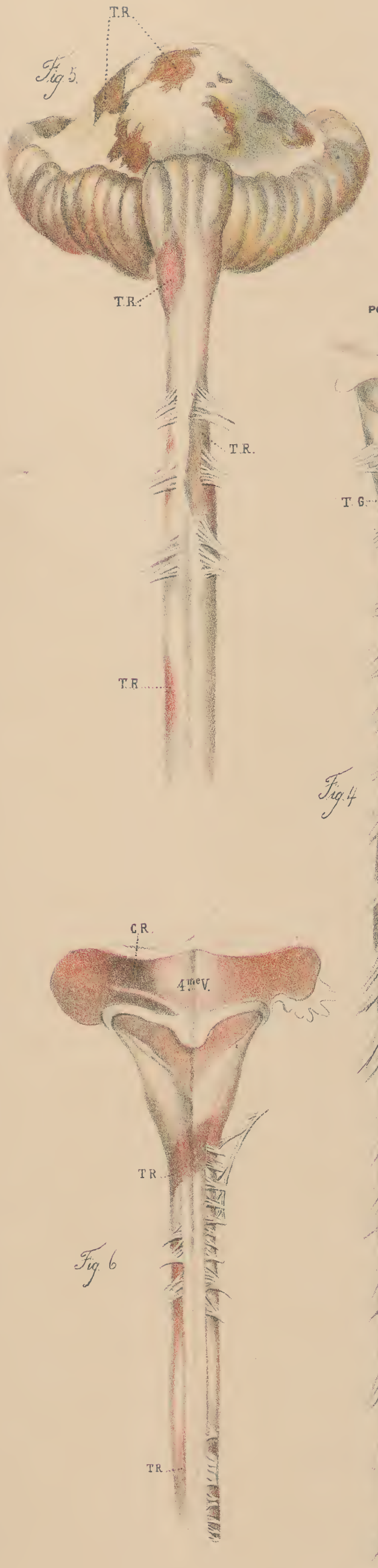

Fig. 6

Fig. 4

Fig. 3

Fig. 1

Fig. 2.

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MYXOMA AND CYSTOMYXOMA OF CEREBELLUM.

SLOW COMPRESSION OF SPINAL MARROW.

Sec. I. *Tab. XIII.*

Fig. 1.

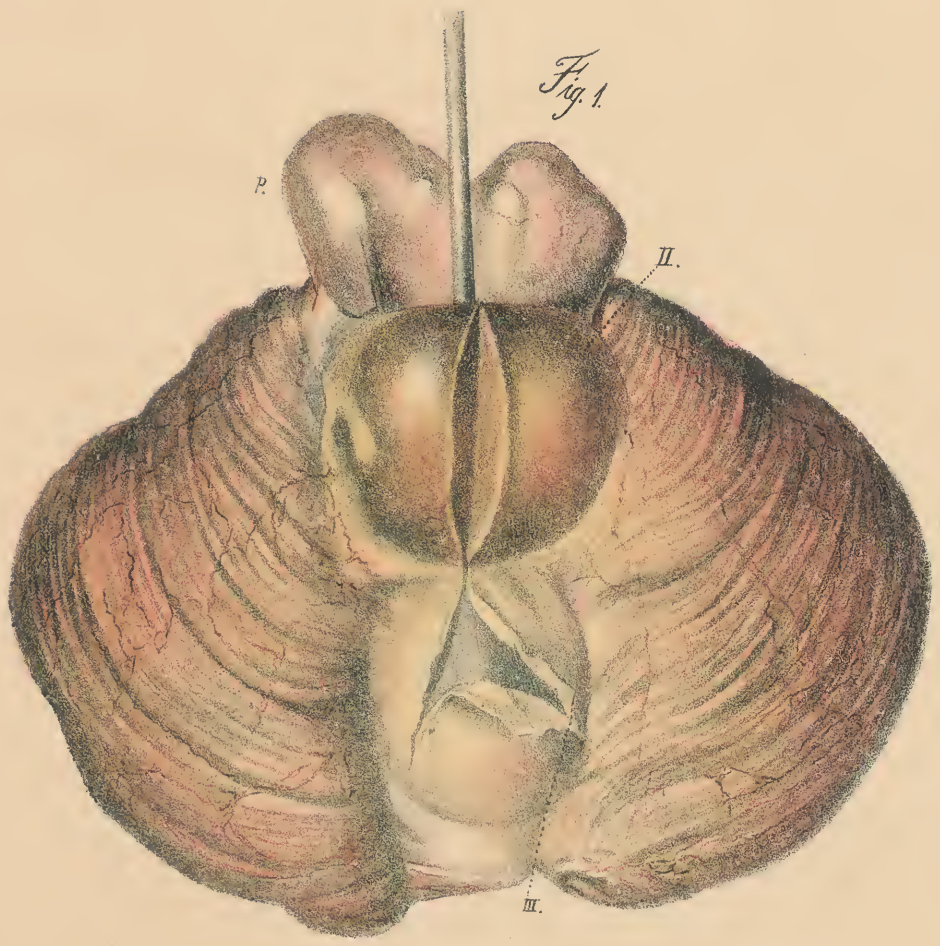

Fig. 2.

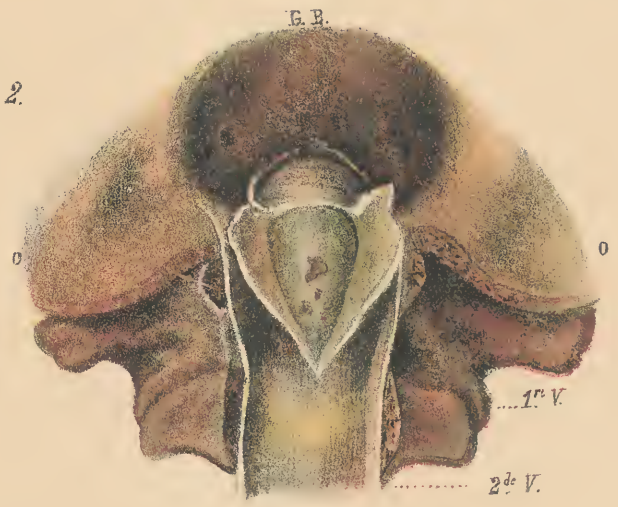

Fig. 3.

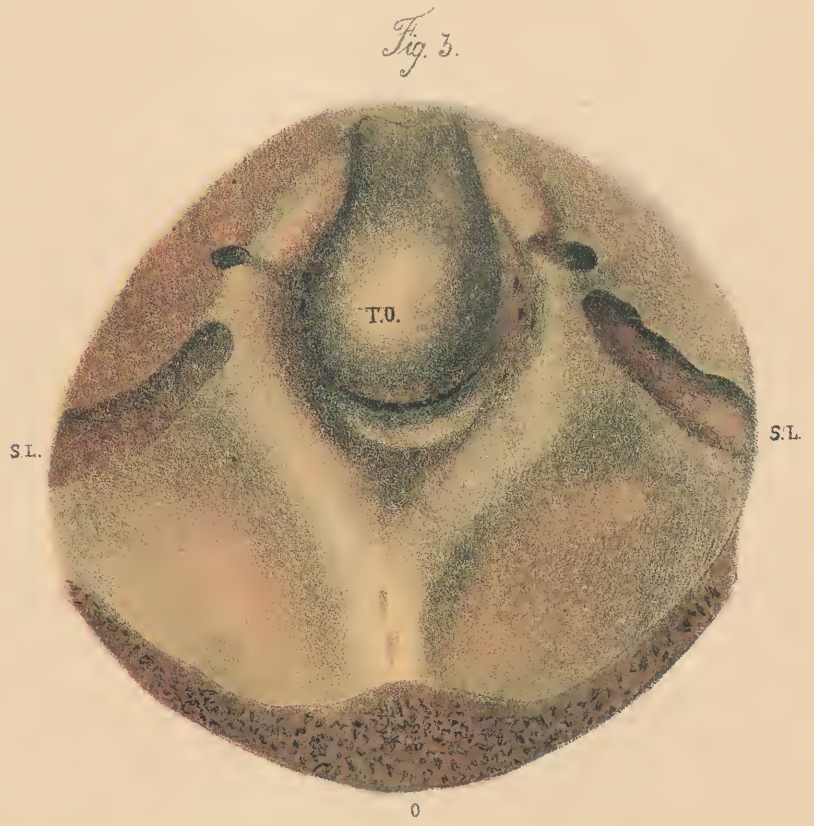

Fig. 4.

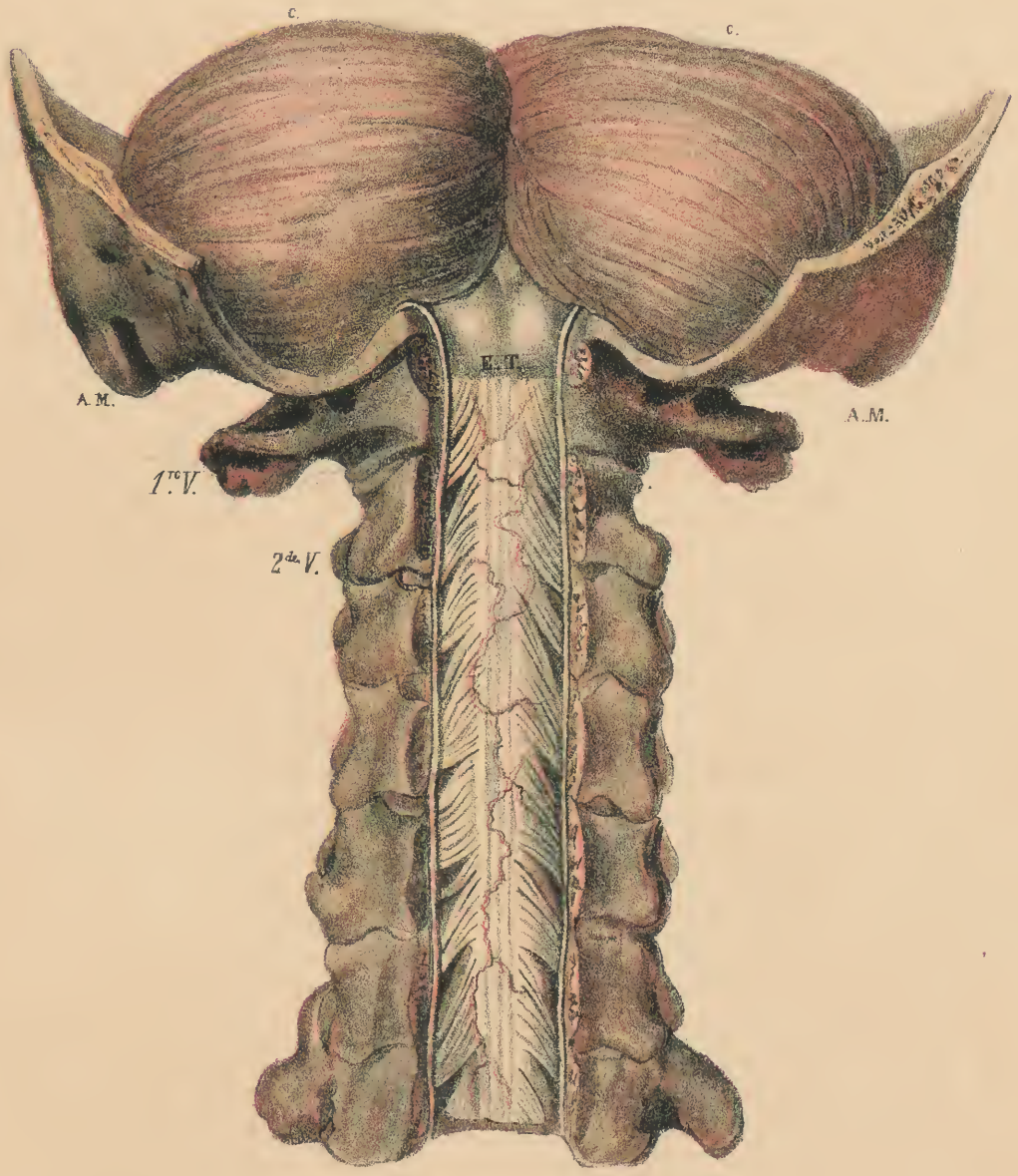

Fig. 5

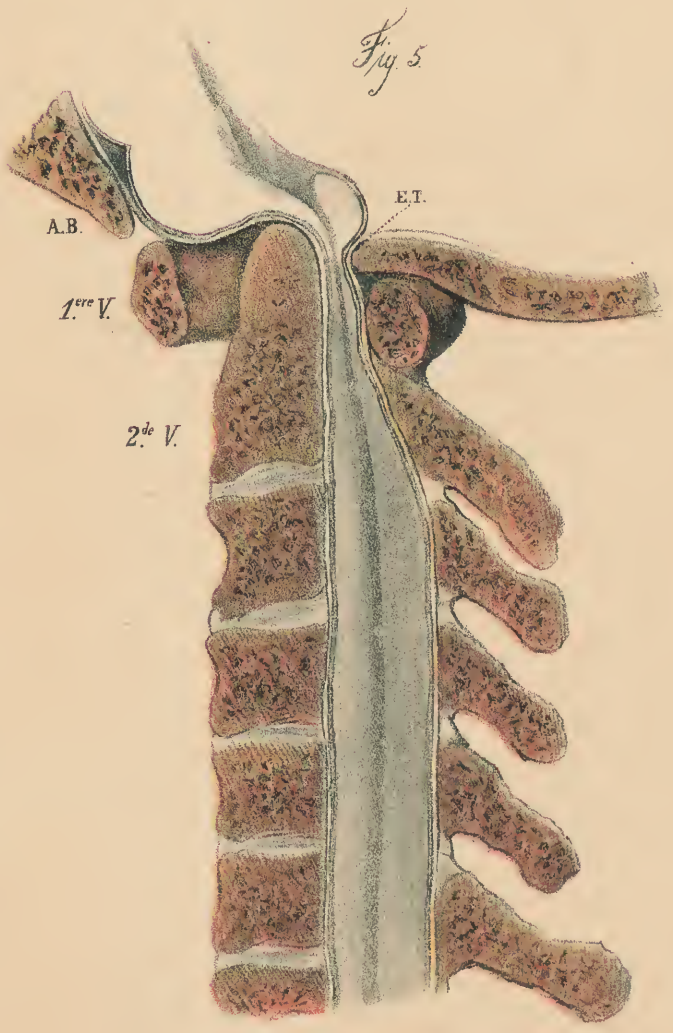

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TABLE XIII.

FIG. 1.—*Myxoma and Cystomyxoma in Cerebellum and Velum Medulare.*

CASE.—Jacob Fuhrman, seven years old. Said to have been a healthy child to his fifth year. Had always an unusually large head. Was two years and a half old before he began to speak. At the age of five years he became unsteady in his gait, and his head leaned much forward as if looking for something on the ground. The unsteadiness of gait gradually increased, and he could not walk without help, and even then he would stumble. He complained only of weakness in the knees and exceeding coldness in the legs. About a year later he became perfectly unable to walk, and when raised up he would drop. Binocular strabismus, of a light degree, was beginning to manifest itself. Slight retention of urine. Complained of no pain. Four months before death he presented the following symptoms: Temperature of skin normal; pulse 92, regular, steady and full; sensorium clear. The most prominent symptom is divergent strabismus of the right eye. He either lies in bed or sits on floor. He moves rather helplessly, along the wall or any piece of furniture. Respiration perfectly normal. Bodily tissues in good condition. With his eyes open, he can stand up. No pain. There is no fever. When the headache comes on he complains of excessive pain in the neck and both legs at the same time. Opthalmoscopic examination showed a slight degree of optic neuritis, some prominence of the papillæ, and considerable turbidity in their contours. No hæmorrhage in fundus occuli. No disturbance of vision. Acute alternating divergent strabismus. Two days before death the pain very intense. Involutary urinary and alvine discharges. Opisthotonus and turning up the eyeballs. Trembling in the limbs, and death.

Post-mortem—(by Prof. V. Recklinghausen). A tumor in the substance of the posterior corpora quadrigemina, which it partly destroyed. Some changes in the dura mater. The pia mater dry. Posteriorly on the basal surface some synechia of the dura and pia mater. A great deal of serum in the ventricles. A section of the superior vermiform process of the cerebellum revealed a good-sized cyst filled with serum; it extended to the top of the fourth ventricle. The tissue surrounding this cyst is very soft and semi-liquid. The tumor is in front of this cyst. It is of gelatinous consistence. The balance of the brain normal.

Pathology of the Case.

There were two periods in this lesion. The first, manifesting symptoms of disturbance of co-ordination, similar to the symptoms existing in certain spinal troubles; and second, symptoms of compression of portions of the brain by a tumor. The optic neuritis suddenly taken place after a fall, distinctly indicated it. The sudden screaming, being a symptom of acute hydrocephalus, is explained by the compression of the venæ galeni, which always causes dropsical effusion.

The ocular symptoms are in relation with the partly destroyed corpora quadrigemina. The alternate diverging strabismus indicates paresis of both oculi motor nerves. Locomotor ataxia can be brought into relation with the trouble of the centers of co-ordination.

FIG. 2.—*Slow Compression of Spinal Cord, Displacement and Caries of the atlas, with localized softening and Sclerosis of the organ by Compression. Gradual atrophy of the organ in the compressed region, followed by ascending and descending Degeneration.*

CASE.—A woman of 68 years.

Symptoms.—Complete right-sided hemiplegia and hemianæsthesia. Involuntary discharges. The hemiplegia set in with a severe chill which has never left her, notwithstanding all means of warming used. To the touch her skin is felt like that of a cold-blooded animal. Suffers very much from a sense of violent oppression. Has a severe pain behind the right ear. In the lower extremities there is often cramp. Some dimness of vision. The cramps often pass from the lower to the upper limbs, and often last for days. Partial epileptic spasms. Dies six months after the attack.

Post-mortem.—No perceptible change in the cerebrum. On the posterior surface of the cerebellum some loss of cortical substance; seemingly an old apoplexy. The atlas is partly dislocated and the spinal marrow immediately below the medulla oblongata is compressed.

Fig. 2 shows the posterior surface of the odontoid process, exposed by cutting through the annular ligament. This ligament is atrophied and degenerated.

Fig. 3 shows the narrowness of the passage between the cranial and spinal cavities, its triangular form and the great projection of the odontoid process (*T. O.*) across the posterior ligament of the *occipito-axoid* articulation.

Fig. 4 shows the furrow resulting from the compression. The left portion of the spinal marrow suffered less than the right, in which the remnant of medullary tissue had undergone gray sclerosis; whilst part of the substance was completely obliterated. In the left portión some white substance was still left. The posterior median columns had undergone gray sclerosis.

Fig. 5 shows the extent of the displacement, and the degree of loss of substance. *E. T.* shows the fissure resulting from that loss.

Pathology of the Case.

Two prominent symptoms: (1.) Complete unilateral hemiplegia and hemianæsthesia. (2.) Extreme algidity of the bodily surface. (The cramps and the partial paralysis of the other side, the dimness of vision, showed a complication with cerebral lesions.) The first indicated that complete motor and sensory paralysis was due to lesions in the anterior and posterior roots on the same side in the spine. The left, which suffered less, showed only a partial paralysis. The extreme coldness of the surface showed destruction of the sympathetic ganglia of the neck and excessive dilation of the blood vessels, causing enormous loss of bodily heat. The dimness of vision shows an extension of the injury to the centra of vision. The cramps were due to reflex irritation of the spasmodic centra in the pons Varoli.

That other agencies besides the above-named may be, or are taking part in the massive serous extravasation in hydrocephalus, both acute and chronic, can not be denied, but the choroid plexus or the tela choroida is certainly the main factor, both in the process of primary effusion and maintenance of the dropsical condition; for, even in the healthy brain, the choroid plexus is ever found in a state of hyperæmia. Added to the hyperæmia is the peculiarity of its covering, which consists of a layer of epithelium, constituting a number of papillæ on the free surface. The immediate connection of the one-layered covering with the vast vascular hyperæmic network, is well calculated to keep up a state of serous effusion on the slightest increase of vascular pressure. Now it has been found that not only is the choroid plexus, but also the whole cranial surface of the pia mater covered with the same epithelial lined vessels. It will be easily understood, when from any cause the vascular papillæ be increased in size and quantity, that an increased quantity of secretion of the liquor cerebro-spinalis will be the consequence. The quantity of such liquid may, to some extent, correspond to the greater or lesser quantity of production of such papillæ upon the plexus, and upon the membrane lining the ventricular walls.

Hæmorrhage, Thrombosis, Embolism and Embolic Infarct of the Vessels of the Pia Mater.

Extravasation of all the constituents of the blood may take place either from the arteries, veins or capillaries of this membrane, or from all the vessels together. Besides traumatic hæmorrhages into the arachnoid sac (caused either by disruption of the veins of the pia mater from the longitudinal sinus, or from rarely occuring rupture of an aneurismal sac of the basilar artery), intercranial hæmorrhages are only caused by the breaking down of the vessels of the cerebral substance proper.

Small hæmorrhagic foci (*Petechiae or Ecchymoses*) occur very frequently in nearly every inflammation of the brain. Rather larger foci are the most frequent here. When a small cerebral artery is obstructed by an embolus, all its branches will become enormously hyperæmic; suffusions will every-where take place, where the vessels are distributed. Endoarteritis of the smallest arterial branches of the basilarartery are liable to produce hæmorrhage like the largest, only in somewhat modified form, and of lesser degree. Besides the numerous changes befalling the walls of the smallest arteries (which become dilated, and constitute either false or true aneurisms), they are converted, according to Virchow, into wide, flabby, thin-walled tubules, in which the arrangements of the histological elements and their three layers can scarcely be recognized. The inner, middle, and external coats are destroyed by a tangible process of proliferation; especially the middle coat, which is otherwise capable to pass from a state of dilatation into that of contraction, and thereby resist the hæmatic pressure- Such a weakened, dilated wall readily yields to any force, and blood passes out of it. Furthermore, the atheromatous process, so clearly set forth by Herzka (*Vortrage, Stuttgart*) as parenchymatous inflammation of the inner walls of the arteries, which become either indurated like cartilage or softened like a jelly by peculiar connective tissue degeneration, gives rise to both massive and ecchymotic hæmorrhages. Even simple hyperæmic inflammation will not seldom produce very massive bleeding. Whilst hæmorrhage from arteries is only produced by solution of continuity of the vascular wall, extravasation of the blood corpuscles from veins and capillaries may take place by diapedesis; that is the exit of the corpuscles bodily through unbroken walls. In this case the extravasation goes on slowly. The quantity of extravasated blood does not depend on the mode of its production, for very

TABLE XIV.

FIGS. 1 TO 6.—*Hæmomyelitis and Descending Paralysis.*

CASE.—I. C. M. Aged 36 years. Was suddenly seized with pain in the neck in the region of the third and fourth cervical vertebræ; gradually extending to the upper and lower extremities, which became paralyzed in succession. The head was turned to the right and could not be moved without intense pain. Pulse regular, respiration likewise; appetite good. Complete paralysis of spinal column, retention of urine and excessive constipation. Complained occasionally of slight headache. Now and then there was a severe pain in the right shoulder. General condition very fair.

Two weeks before death the bed-sore on his sacrum became gangrenous, and very fetid, and he was seized with violent fever and vomited blood. Died in collapse.

Post-mortem. — Brain normal. Membranes of brain same. Spine: Its membranes normal with exception of a spot in the lower portion of the fibrous sheath of the last sacral vertebra, which was affected by gangrene. (From caries of vertebra.) Opening the dura mater spinalis, a large quantity of serum was found in the sub-arachnoidal space in the region between the medulla oblongata and the cervical medulla. (It explains why the head turned to the left.) Between the origins of the fourth, fifth and sixth pair of cervical roots of the spinal nerves, a tumor, about an inch long and a half wide (*F. A. P.* Fig. 1.), was discovered, the posterior cords of the spinal marrow, to the left, were infiltrated with blood and raised. They were separated from the adjoining cords, but were not broken. The hæmorrhagic focus extending, externally, from the anterior to the posterior roots of the named nerves, lifted and separated them; and gave them a purplish color. There was no solution of continuity. The inferior portion of the *cauda equinæ* presented traces of an old hæmorrhage (Fig. 2.). Anteriorly (Fig. 3.) the hæmorrhagic portion was more prominent (*F. A. A.*) than the posterior (*F. A. P.*). Here the fascicles were torn, and some were obliterated. The fibres of the anterior roots were here reduced to their neurolema.

A longitudinal incision in the spinal marrow posteriorly showed that there was an extensive hæmorrhage in the center, of a very recent date. Dividing the median commissure, and widely separating the two halves, the whole portion of the gray spinal substance was found perfectly infiltrated with blood (4, 5), from the origin of the medulla oblongata (*M. O.* Fig. 4.) to the lower expansion of the spinal cord (*M. O.* Fig. 4.). The upper portion was more infiltrated than the lower. (*F. A. O.* shows the extent of the hæmorrhage in its several aspects.)

limited bleeding may occur by rupture of a vein, and very extensive by diapedesis.

In multiple punctiform hæmorrhage, the whole surface of the cortex is spread over with ecchymotic spots. In circumscribed, they are confined to different localities. The aspect of the brain following immediately such punctiform hæmorrhage, by whatever cause produced, is nearly alike, that is: the region around the bleeding vessels is penetrated by a number of hæmorrhagic points, for a quarter of an inch to an inch. The number of the points increase in quantity, the closer they are to the focus. In the center itself the spots are very close, and appear, to the naked eye, as one large bleeding mass. The partly compressed and partly displaced portions of the brain, by the hæmorrhage, bear a reddish or pinkish color; farther away they have a more yellowish tinge. (*See Secton I, Tab. VII, Figs. 1, 2.*) The alterations following such miliary hæmorrhages are: finely-fibred conglomerate produced in the center of each focus, where the *vessels* were situated, fibrinous capsules enclosing each little drop of blood. These latter, seemingly secondary products in the periphery of the focus, consist of the extravasated fibrin from the blood vessel; and its coagulation is due to fibro-plastic zymogen found in the blood corpuscles. The farther changes are as follows: yellow softening, purulent degeneration and decay, direct regeneration of tissue. Their histological changes will be described in the microscopic portion of this work.

The agencies capable of producing minute hæmorrhages are also productive of massive extravasations, differing only in degree of effect. The cases described in Tab. VII., Figs. 2, 3, 4, 5, and Tab. VIII, Figs. 2, 3, are examples of massive hæmorrhages. Larger branches of the Sylvian artery, leading to the corpus striatum, were ruptured, and the extravasated blood spreading with great force, displaced the cerebral structures in all directions. Outside, it penetrated into the center of Vieussens. Internally the thalamus opticus was driven in, the corpus striatum raised and perforated in a number of places, and the ventricles partly filled with blood. The space required for such a quantity of blood was gained at the expense of the cerebro-spinal liquid, which it displaced, of the contents of the blood-vessels which was driven out of them, and the vessels compressed. By removing the dura mater from the cerebral surface, the sulci were found effaced, the Gyri flattened and the veins perfectly empty.

Usually death follows such extensive hæmorrhages. Rarely recovery takes place. When it does happen, the extravasated blood is enclosed in a fibrinous capsule (a sort of demarcation), derived from the same source as the blood itself. This capsule generally undergoes the same metamorphosis as those of the smaller extravasates. The ulterior changes taking place in the reconstructed tissue vary, greatly, with the subsequent conditions of the body, etc. The extravasated blood, in whatever quantity, provided the person lives for a sufficient length of time after the apoplectic attack, may undergo several changes. A portion of its hæmatin may dissolve and diffuse itself by imbibition into the surrounding tissue. Another portion may be absorbed in the perivascular lymph-spaces and by a number of peculiar large cells capable of taking up and retaining quite a number of blood corpuscles. Another may pass the pigmentary metamorphosis, and produce copious deposits of yellow pigment granules and hæmatoidin crystals in the cicatricial tissue. Still another portion may be converted into connective tissue and unite the capsular walls and form a closed sac. This sac may either become gradually atrophied, shrink away and form a cicatricial stripe, or it may gain in thickness, by secondary deposits of fibrin, and form an *"apoplectic cyst."* Such a cyst is filled with a yellowish or colorless liquid, in which float the organized remnants of the extravasate. As a general thing, surface hæmorrhages form cicatrices.

Inflammation of the cerebral meningiæ is due to various causes. These may be either of a traumatic nature or idiopathic, that is, from no local injury produced mechanically, etc. In acute forms of the disease, the symptoms, although not always constant in their grouping, are, nevertheless, sufficiently distinct to indicate the nature of the disease; especially when the inflammation is either extending over a large portion of the membrane, or diffusely spread in smaller foci over the whole or the greatest portion of it.

The anatomical peculiarities of the membranes help to throw light upon the differential state of disease of each and assist in their differential diagnosis. Forstner (Archiv of psychophysiology) gives a synoptical description of the symptoms of hæmorrhagic pachymeningitis. "Tuberculous persons may be attacked by inflammation of the dura mater. The hæmorrhage produced by it causes a sudden motor irritation (if the person affected is paralytic or a hard drinker); restlessness and delirium ensue. Then follow congestion of the face, profuse perspiration, and great increase of frequency of pulse. Gradually the exitatory phenomena subside, the pulse grows slower and weaker, becomes sometimes very irregular. The same is the case with the respiration; drowsiness and coma ensue; the latter condition lasting, sometime, for days. Paleness of face generally exists shortly before death."

"In delirium tremens, it assumes a protracted character, with occasional intermissions of the delirious condition." In œdema of the pia mater peculiar to drunkards, the symptoms are similar to those described. Occasionally the following symptoms will be manifested in pachymeningitis, viz.: a sudden attack, or several attacks, of epileptiform convulsions, alternately affecting one, then the other half of the body. In the intervals between the attacks a state of coma exists. This generally proves fatal. In the rarest cases has it the form of apoplexy. A nearly constant symptom in hæmorrhagic pachymeningitis is a very high temperature of the body, often reaching 105° Fahr. and upward.

"Besides the remittent character of the coma, there exists single or double *intraocular infiltration*, caused by the extravasation of blood in the sheath of the optic nerve, deviation conjuguee and one-sided nystagmus. The pupils will often alternately dilate and contract. Hemiplegia occurs very seldom, unless it be in very extensive hæmorrhage, the motor paralysis being either on the same side or passing to the opposite one. Paresis of some portions of the body, or spreading to a number of muscles over the whole body, is the most frequent occurrence. Contractions of paralyzed limbs will occasionally take place. Graver disturbances of sensation are very rare in the disease. When stiffness of the neck and paralysis of the basal nerves exist, it shows that the disease is not pachymeningitis." Besides the symptoms of meningeal disease, described in reports of the diseases in this section, the following, taken here and there from cases reported in current medical literature, will show the variety of symptoms manifested under different circumstances in these diseases.

Magnan: "Two cases of hæmorrhagic pachymeningitis. 1. General paralysis, epileptiform attacks, followed by left-sided hemiplegia; afterward followed by right-sided hemiplegia and aphasia. Nature of lesion; double hæmatoma of dura, compressing hemispheres. 2. Chronic alcoholism; right-sided hæmorrhagic pachymeningitis. Suppuration in hæmatoma. Persistent headache, left-sided hemiplegia (*Gazette Medical, No. 31*)." Friedreich describes a case of chronic arachnitis in *Norsk Magazine f. Laegaerd, No. 3, Vol. 9, Page 48*: "Insolation, loss of memory, gradually growing idiocy, both lower limbs paretic. After a drunken orgie he is attacked with severe headache, severe vomiting, and from date falls into a state of gradual general atrophy, with uncountable pulse, intercurrent facial paralysis, binocular divergent strabismus, ptosis, epileptiform convulsions and death. Duration of the diseases eight years."

"*Post-mortem.*—Dura mater loosely adherent to cranium. The whole arachnoid upon convex surface of cerebrum opaque, thickened, and very tough. Abundance of serum in the sub-arachnoidal space. The nerves emerging upon basal surface partly sclerotic, pia mater, with little exception, normal, *Granuloma* in left lenticular nucleus." (*F. Sorel, in "Receuil de med. milit. No. 3," page 269*, de-

Fig. 1.

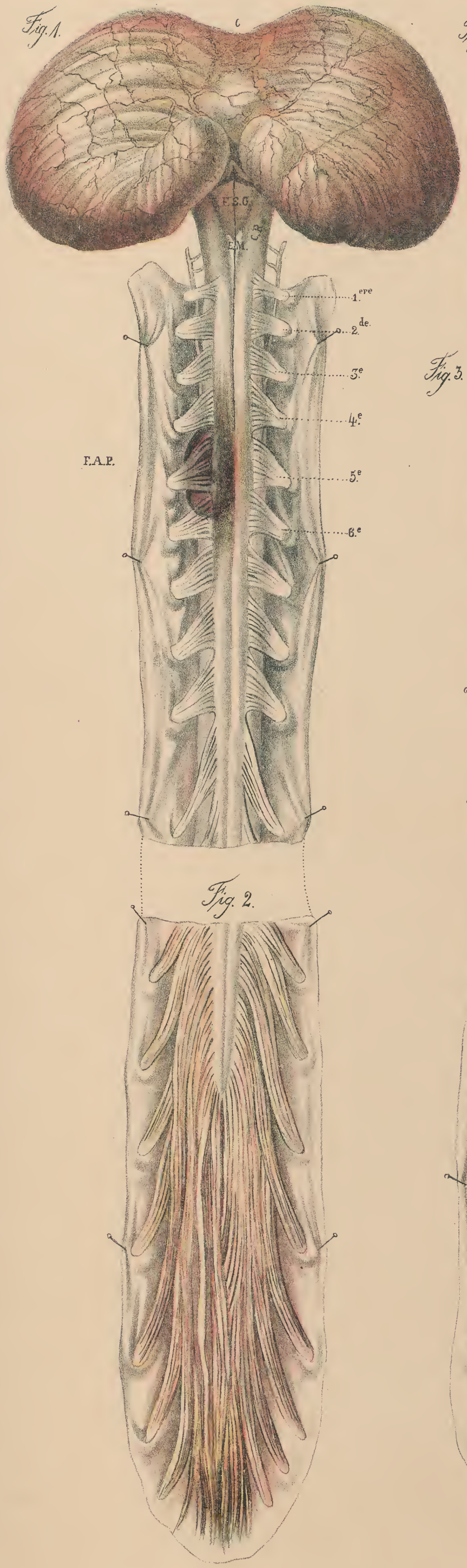


Fig. 2.

Fig. 6.

Fig. 3.

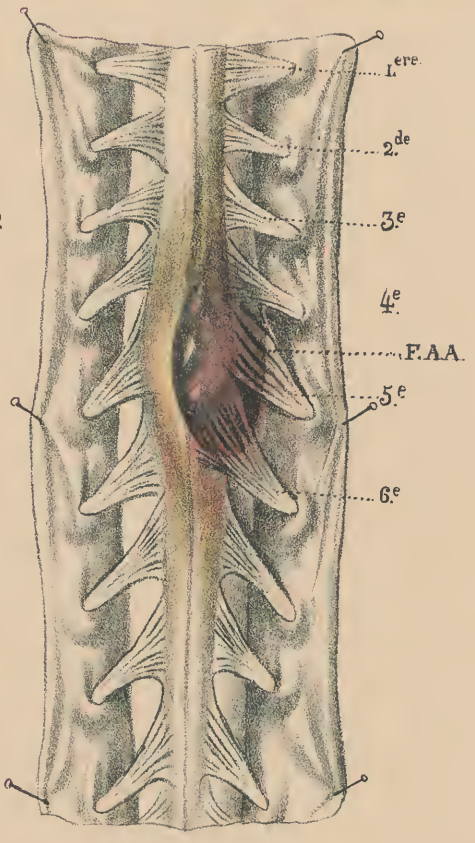


Fig. 5.

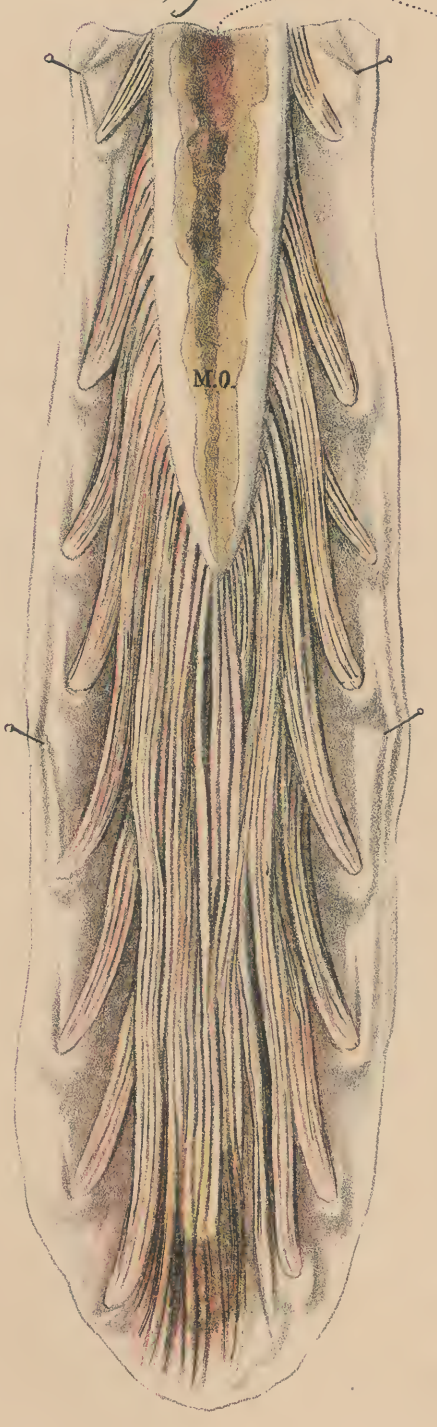
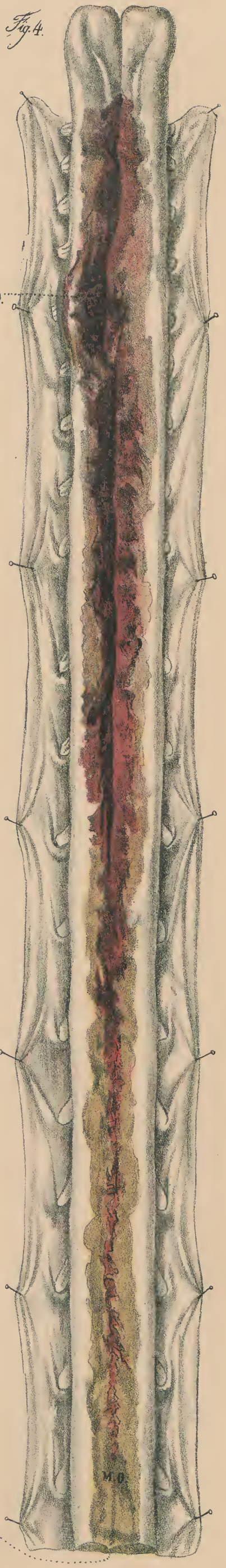


Fig. 4.



Sec. I. *Tab. XV.*

ACUTE ASCENDING PARALYSIS.
HÆMORRHAGIC MENINGO-MYELITIS.
COMPLETE TRANSVERSE ATROPHY.

Fig. 1.

Fig. 2

Fig. 3.

Fig. 5.

Fig. 5.

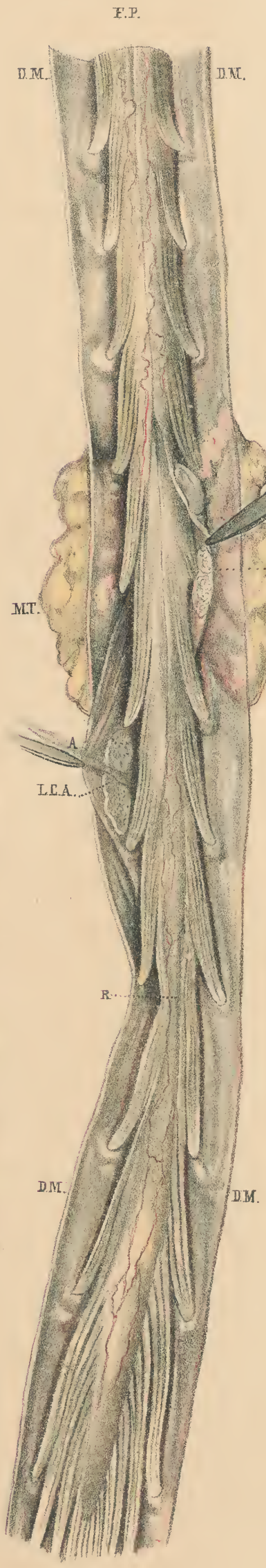


Fig. 4.

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Table XV.

Fig. 1.—*Acute Ascending Paralysis.*

Case.—A man of forty years. In ordinary good health. Four years before his death contracted syphilis, was treated and considered cured. Three years afterward he was taken with a sense of fatigue and weakness in the lower extremities, from which he soon recovered. Subsequently, paresis of the muscles of the arms, neck and abdomen set in, with very painful and difficult swallowing and mastication. Then occurred total extinction of cutaneous and patellar reflex irritation. Faradic currents had no effect upon the muscles of any of the extremities, galvanic currents produced some tetanic rigidity of the muscles of the legs. Direct irritation of the ulnar and radial nerves produced some slight twitching. Shortly afterward died from exhaustion.

Post-mortem.—Acute myelitis, especially of the motor conductors (pyramids and the lateral cords), of the anterior gray substance, throughout the whole length of the marrow, and of the medulla oblongata, up to the level of the nucleus of the fifth nerve. In the gray matter of the anterior cornua, the ganglionic cells were all bloated, full of coarse granules, and perforated by many vacuola. The posterior cords and roots were far less injured. The meningiæ suffered but little. The muscular tissue of the lower extremities degenerated.

Fig. 1. Anterior aspect (*F. A.*). Fig. 2. Posterior aspect (*F. P.*). Fig. 3. Section of spinal marrow (*C. T.*) degeneration.

Fig. 4. *Hæmorrhagic meningo-myelitis with ascending neuritis.* Complete paralysis of inferior extremities. Red softening of anterior cords; atrophy of gray substance around the central canal. Enormous hæmorrhagic focus in lumbar region.

Figs. 5, 5.—*Complete Transverse Atrophy from Slow Compression of the Spinal Marrow, Vertebral Caries.*

Case.—L., thirty-five years old. Symptoms: Considerable lateral curvature in lower dorsal region, involving several vertebræ. Complete motor and sensory paralysis of lower extremities. Intermittent incontinence of urine. Lower extremities flexed and rigid. Occasionally severe spasmodic attacks in those muscles. Bed-sores and gangrene. Acute purulent pleuritis; death.

Post-mortem.—A wide, suppurating sinus, forming a communication between the spinal canal in the lower dorsal region and the left pleural cavity. A voluminous, soft, yellowish-white mass was found to exist in the lower dorsal region, inside the spinal dura mater, which caused a gradual pressure upon the spinal marrow, and nearly obliterated the central gray substance to the extent of an inch (5). The two lower dorsal vertebræ were affected with caries, and presented a suppurating surface where the sinus began. The spinal marrow was constricted for nearly an inch in length (*M. T.*). A number of cartilaginous plates were found adherent to the dural surface of the arachnoid (*L. C. A. C. A.*). On the surface of the pia mater they were loose. (*D. M.* Dura mater. *R.* Constricted spinal marrow.

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scribes symptoms in meningo-cephalic tuberculosis, "cortical lesion confined, on the left, to motor Zone, *associated monoplegia of the opposite limbs.*"–*Wengler in deutsch Archiv. fuer clin. med., Vol. 26, Page 179,* *Reports*: "Tubercular meningitis associated with aphasia, right-sided convulsions, immovable pupils of both eyes, vomiting." The same author in *Wiener Wochenschrift*, No. 9, describes basilar meningitis: "Excessive stiffness of neck, difficulty of deglutition, protrusion of left bulb with subsequent profuse suppuration of the vitrous body, fever and delirium, death. Lesion: purulent infiltration of pia mater, basilar portion, infiltration of all ventricles with serum." *Hodgeson in British med. journal, Sep. 18, 1875*, tells of a case of meningial apoplexy as follows: "Sudden attack of intense pain in the back of the head and neck, becomes numb all over the body very quickly. Barely any other symptoms. On the third day he is furiously delirious; gradual paralysis of left arm and leg; speedy death. Post-mortem: Exceedingly hyperæmic brain, in the right sub-arachnoidal space of the medulla oblongata very extensive hæmorrhage, cerebral arteries atheromatous, acute hydrocephalus."

Pathological Localization in the Cerebral Peduncles.

When a pathological center is situated in the anterior portion of a cerebral peduncle, it very rarely fails to affect the origin of the third pair. Should the root of that nerve be involved, there will be an alternate paralysis of the third pair and a hemiplegia of the whole opposite side of the body. A unilateral, but posterior lesion of the peduncle may give rise to alternate paralysis of the oculo motor communis with hemianæsthesia of the opposite side of the face and the limbs. The optic fascicle and the root of the pathetic, surround the peduncle at this point, and when a lesion exists there, the following symptoms will be manifested: hemiopia, paralysis of the superior oblique, etc. A lesion in the superior posterior portion of the peduncle, the *tegmentum cruris*, and immediately behind the optic thalamus will be manifested by hemichorea. Some peduncular affections give rise to disturbance of co-ordination. There is never manifested, in any of those lesions, any disturbance of the intellect.

Localization in the Pons.

The gray matter of the pons varoli forms a square band, situated in its posterior portion, and corresponds to the floor of the fourth ventricle. It is here formed by the nuclei of the superior facial, trigeminus, and the external motor oculi. Above, it surrounds the aqueduct of Sylvius like a sheath, and contains the nuclei of the common motor oculi and the pathetic. A number of smaller nervous centra exist here, which have as yet not been clearly defined.

Where the peduncles emerge, the *locus niger* of Soemmering is situated. It is, no doubt, a functional center which has not been sufficiently studied as yet. The pontine nerve-fibres form vertical and horizontal bundles. The latter constitute the *pons* proper. They occupy the anterior surface of the organ, and seem to be the divergent fibers of the middle cerebral peduncle. Some of these pass into the body of the pons, between the vertical bundles. According to Hillairet, a lesion in these bundles produces a sort of ataxia and titubation in the gait. There are some antero-posterior and slightly oblique fibers between which nerve-nuclei are situated. The vertical bundles form two groups: (1.) Median and anterior, divided in secondary fascicles, which enter the pyramids and are voluntary motor fibres. (2.) Lateral and posterior; they are centripital or sensory fibers, originating in the medulla oblongata, and are directed towards the encephalic ganglia. Mr. Couty, after many observations, came to the conclusion that *sensory* disturbances are most frequent in lesions of the posterior layers of the pons, whilst anterior and median lesions cause motor disturbances. Two prominent symptoms are chiefly characteristic of diseases of the pons. (1.) *Alternate hemiplegia and hemianæsthesia.* (2.) Disturbance of the sense of hearing and smell.

Alternate hemiplegia or hemianæsthesia, since its description by M. Gubler, has been considered as a symptom of pontine lesions; such are: right hemiplegia of the face and left hemiplegia of the limbs, etc. Dr. Siegerson described two other varieties of alternate paralysis: (1.) Double alternate, complete paralysis in the form of an X; for instance, the two opposite sides of the face and the two opposite sides of the body are affected. (2.) Incomplete double alternate paralysis, in the form of a V; for instance, when only the two sides of the face are paralyzed; or in the form of a Y when two sides of the face and one side of the body, and in the form of Λ when two sides of the body and one side of the face.

Another symptomatic character of lesions of the pons is: *generally co-existing derangement of the sense of hearing and of taste*, with the anæsthesia or paralysis. Cerebral hemianæsthesia is associated with loss of sight and olfaction, as well as derangement in the other organs of sense. In protuberantial lesions and in lesions of the medulla oblongata, the senses of sight and olfaction are undisturbed, only the hearing and the taste are affected. One negative symptom, that is, there never exists any strictly mental disturbance in purely pontine lesions. The pons may be divided in three different regions, for clinical purposes.

(1.) A vertical plane, median antero-posterior.

(2.) A horizontal, in the apparent origin of the trigeminus.

(3.) A transverse and vertical, perpendicular to the middle of the horizontal plane.

(A.) Lesions situated in one side of the median antero-posterior plane and extending into the entire half of the pons produce semi-lateral derangements of the opposite sides of the face and of the body, when situated below the horizontal plane; when situated above that plane, and of the same extent as the above, alternate hemianæsthesia and hemiplegia is the consequence. These may be complicated with derangement of hearing and taste.

When lateral lesions are of slight extent, there will be only hemiplegia, when situated in front; when situated posteriorly, only hemianæsthesia.

(B.) Lesions situated above the horizontal plane will produce hemiplegia of the opposite side, when only the motor bundles are involved. An alternate paralysis of the pathetic and of the motor oculi communis may also take place.

When situated behind, that is, among the sensory bundles, only hemianæsthesia will be produced.

Above the horizontal plane, lateral lesions occupying all that half of the pons, produce alternate hemiplegia and hemianæsthesia. When the extent is small, and in front, only alternate hemiplegia will follow; if situated posteriorly, alternate hemianæsthesia will be the consequence.

(C.) In front of the vertical and transverse planes, and perpendicular to the horizontal, there will be chiefly motor disturbances; when posteriorly situated, chiefly sensory derangement will follow.

(D.) Lesions situated in the median line and extending on both sides, will produce disturbances in both sides of the body.

Pathological Localization in the Medulla Oblongata.

Lesions of the medulla oblongata situated in the nuclei of the gray matter, always produce a group of symptoms, which may be qualified as cephalic, or cardiac and respiratory derangements. The best known cephalic symptoms are those which are the result of lesions of the inferior nuclei, situated in the floor of the fourth ventricle; these are of the spinal accessory, hypoglossal, and inferior facial. They constitute the so-called labio-glosso-laryngeal paralysis. There are two forms of this lesion, one described by Duchenne in 1860, as consisting, essentially, of a paralytic affection gradually invading the *muscles of the tongue, the soft palate,* the *orbicularis oris.* At first there is disturbance of speech, difficulty of swallowing, afterward, difficulty of breathing. For the progressive atrophy, affecting the nuclei of the spinal accessory,

TABLE XVI.

CHRONIC CENTRAL MYELITIS, BELOW THE LUMBAR REGION, WITH COMPLETE PARALYSIS OF LOWER EXTREMITIES.

CASE.—Secondary myelitis from gradual pressure upon the lower lumbar portion of the spinal marrow. There was complete loss of motion and sensation in lower extremities. No *girdling sensation* in abdominal region. Perfect freedom of motion and undisturbed sensation above the lower dorsal vertebra. Death from exhaustion.

Post-mortem.—Synechia of dura and pia mater. Dura mater much injected and swollen. Central canal obliterated, central gray substance highly atrophied, portions semi-liquid, and remnants of ganglionic cells and glia corpuscles completely degenerated. A state of anæmia of the spinal marrow below the region of the last dorsal vertebra seems to have existed for many years. Above that region the whole organ appeared perfectly normal.

Fig. 1. Posterior aspect. (*D. M.*) Dura mater, its vessels widened, and the dural substance slightly infiltrated. (*P.*) Pia mater, anæmic and pale.

Fig. 2. Anterior aspect, shows adhesion of membranes, and anæmic pia.

Fig. 3. Longitudinal section showing the grayish, yellow, soft, degenerated tissue, and obliteration of its peculiar structure.

FIGS. 4, 5.—SYRINGO-MYELITIS.

4. Anterior. 5. Posterior surface.

The anterior surface shows that the spinal marrow is swollen and enlarged in certain portions, whilst it is of normal volume in others.

The post-mortem showed that the spinal dura mater was highly vascular and the pia mater opaque and infiltrated with a grayish serum. The opacity was greater behind than in front. The dilated portion existing in the marrow itself was due to a considerable dilatation of the central canal, which gradually narrowed down to a normal width below the sixth cervical vertebra. The dilated central canal was surrounded by a layer of transparent, fairly consistent connective tissue, about 2 m.m. thick.

The dilatation of the canal was rather eccentric, for it extended into the posterior cords; yet it was originally proceeding from the central portion, and was only a part of that cavity; for in *its anterior* wall distinct quantities of *cylindrical epithelium* could be found. Inside of this cavity existed quite a quantity of a peculiar substance, perfectly different from the spinal marrow substance. It was of a gelatinous consistence, and was made of net-work of fine fibres, containing a quantity of small granules. Not a trace of a vessel or nerve was there, but the mass resembled embryonal structure from which the medullary tissue is produced. The whole abnormal condition of the spinal marrow showed its intimate relation to hydrocephalus; the symptoms during the life of the patient were identical with those of that lesion.

FIGS. 6, 7, 8.—*Hydromyelus and Spina Bifida.*

Fig. 6 represents the medulla oblongata of a three-year-old child, who died from hydromyelus and spina bifida. It shows a vertical section of the cerebro-spinal canal, and the arachnoidal spaces filled with a thick, greenish pus (*P. P*). The cerebellum (*C.*) is remarkably small. Fig. 8 shows the details of the lumbar tumor (*T. L.*), and the spinal marrow dipping into its walls where it is lost. The roots of the spinal nerves emerge from the tumor and form the *sacral plexus* and its ganglia (Fig. 7), (*G. L*) lumbar ganglia, (*G. S.*) sacral ganglia.

Fig. 9 shows the anterior aspect of the modified spinal canal.

Fig. 10, the posterior surface. They both show how far the individual vertebræ were modified and abnormally placed, and in what manner the closure of the spinal canal was prevented.

Pathological Anatomy of Spina Bifida.

Pouchet in Revue scientifique (1875) expresses himself concerning it in this manner: "There exists a malformation known under the name of *spina bifida*, in which the two first, parallel, dorsal crests of the blastoderm do not unite on the median line. These normally join and form the central canal of the spinal marrow. The cells which line this canal originally partake of the superficial elements of the embryo; they are in immediate continuity with the cells constituting the epidermis. Only after the closure of the canal do they assume their own proper histological character. When the canal remains open, what becomes of those cells? When they do not line the medullary cavity, they remain in contact with the *amniotic* liquid where the rest of the embryonal body is plunged." —"Had the canal been closed and the cerebro-spinal axis formed, the superficial cells would have become the epithelium of the ependyma, and the internal the myelocytes, from which the pia mater on one hand, and the cerebro-spinal axial nerves on the other, would have developed."—(It is now conceded on all hands that spina bifida is due to a stoppage of embryonal development at a certain period of its existence.—F. Tourneux and E. Martin, *Journal de l'anatomie*, 1881, Page 12.) (Mr. Dareste, after a number of observations, has classified all spinal fissures under four heads: 1. In which the medullary lamella, the origin of the medullary tube and the cerebro-spinal axis, does not close up. It remains at the bottom of the medullary fissure and retains its communication with the serous blastoderm. It becomes the starting point of spina bifida, in which no pocket exists. 2. The medullary lamella forms a tube, but developes later than normally. The parts of the walls do not unite, but remain separate; but unite with the *serous derma*. 3. The medullary lamella remains as in case 2, but separate from the serous layer. The dorsal lamellæ do not approach each other, the osseous elements remain separate and the fissure has the width of this gap. 4. The medullary lamella is completely developed, but the cerebro-spinal axis may be compressed by the amnios.

The skin and the meningiæ close up in the median line, but the bony canal remains open."

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the hypoglossus and the lower facial gradually extend to the nucleus of the pneumo-gastric, and paralyzing the respiratory apparatus, produce attacks of fainting and smothering, which, in the long run, suffocate the patient.

The second form of this lesion is: the *bulbo-spinal*, or *progressive muscular atrophy;* either beginning in the medulla oblongata and extending to the spinal marrow, or commencing in the latter and ascending into the former. The peculiar character of the symptoms in the second form is: first, that there is always definite muscular atrophy; second, that the atrophy and the paralysis are progressive.

In descending paralysis, it begins to manifest itself, after the bulbar symptoms, in the muscles of the neck and the upper muscles of the upper extremities.

In ascending paralysis, the affection is manifested by progressive muscular atrophy of the extremities, afterwards there is gradual increase of impediment to speech and deglutition. Charcot, Geoffray and Gombault have proven that in either case there is progressive ascending or descending degeneracy of the ganglionic cells. The superior portions of the medulla oblongata, corresponding to the nuclei of the motor nerves of the eyes, may become localized centers of lesion in their several cellular groups. They may furnish explanation of a variety of limited paralyses of some muscles of the eyes and eyelids. There must certainly exist centrally located lesions in the nuclei of the mixed and sensory nerves, which may produce definite groups of spmptoms, but which have as yet not been defined.

Lesions Located in the White Substance of the Medulla Oblongata.

Encephalic and medullary sclerosis often find place in the transmissive organs (the white ascending and descending fascicles) in the medulla oblongata. These bundles may be singly or collectively affected, or in spots, in several localities.

A. Lateral Amyatrophic Sclerosis. Bulbar symptoms may be superadded to the spinal in most cases of the kind; they constitute the second phase or last stage of the spinal disease. The progressive muscular *paresis* passing into *paralysis* of the muscles of the tongue, lips and pharynx. The nature of these lesions may consist of either: degeneracy of the nuclear cells in the inferior portion of the medulla oblongata; pyramidal sclerosis, that is a chronic inflammatory condition, passing from the sides to the front; or the atrophy of the glosso-labio-laryngeal group of muscles.

B. Disseminated Sclerosis may produce the same morbid phenomena as these.

C. Descending Degeneracy from hæmorrhagic, or necrobiotic foci of the encephalon, and occupying the anterior pyramids.

D. Diffuse affections of the spinal medulla may extend the morbid process into the medulla oblongata, for instance: hypertrophic cervical pachymeningitis, dementia paralytica, etc.

The medulla oblongata is rarely the seat of *focal* lesions. Still, a sufficient number of cases is reported to justify the belief that acute focal hæmorrhages may suddenly take place in the organ. For instance the following cases: ("Lichtheim, two cases of apoplectiform bulbar paralysis.—Berlin, Klin. Wochenschrift No. 12. 2, deutsches Archiv für Klin. Medicin, Vol. XVIII, p. 593.") ("Fischl apoplectiform bulbar paralysis, Prag. medic. Wochenshrift No. 5, 6, 7.") ("Leyden, Archiv für Psych, VII, p. 44, two cases of focal lesions.") (Israel, deutsche Zeitschrift f. pract. Med., No. 35, 36.") ("Hallopeau, These de Paris 152 pp. des paralyses bulbaires, a hæmorrhagic and necrotiotic focus.") ("Habershon, glosso-laryngeal paralysis, Guy's Hosp. Rep. XX, p. 334, a softened hæmorrhagic focus in the right olivary body.") Many more can be cited.

The patients may be suddenly seized with paralysis and manifest all the symptoms of recent spinal paralysis. Such sudden attacks are liable soon to recover, or at least gradually to be mitigated, provided the patients are able to pass through the acute inflammatory process.

In case of death, post-mortem in such cases will be found: A clot or clots which obliterate the circulation in either of the trunks of the two vertebral arteries, a few lines perhaps from their point of junction with the basilar. Duret explains it in this manner (Etude General). The anterior spinal arteries originate in the vertebral on a level with the occluded vessels, and, as the blood cannot pass into these branches, there may form a sudden anæmia in the inferior nuclei of the medulla oblongata (hypoglossus, spinal accessory, inferior facial) which these arteries supply. Should the clot ascend higher, or should a thrombus be formed in the inferior portion of the basilar artery, sudden death will take place; then from here are also derived the nutrient arteries of the nucleus of the pneumo-gastric, the paralysis of which produces sudden death.

Hayem describes ten cases where death was produced by the occlusion of the basilar. When the clot occupies the superior portion of the basilar artery, paralysis of the muscles of the upper portion of the face or of the eyes may be looked for, then the nuclei of the oculo motor and the superior facial derive their nutrition from that portion of the trunk.

Such bulbar lesions produced by vascular emboli or thrombi are always associated with a certain degree of paralysis and anæsthesia in one limb, in two limbs of the opposite side, or in all four limbs. For nearly all the branches supplying the bulbar nuclei, the motor and sensory fascicles of the organ may become involved.

Traumatic Lesions Located in the Medulla Oblongata.

A violent blow upon the skull, a fall upon the head, may cause sudden death. The cause of the sudden death is not so much due to injuries of the cerebrum or to other important cerebral centra, as to that of an injury or even a shock to a very limited locality in the medulla oblongata, that is, the locality of the nucleus of the pneumo-gastric, at the apex of the gray substance of the fourth ventricle.

(Continued in the neurological part of this work.)

DISEASES OF THE CEREBRO-SPINAL AXIS AND OF ITS MEMBRANES.

CHRONIC CENTRAL MYELITIS.
SYRINGOMYELUS.
HYDROMYELUS, SPINA BIFIDA.

Sec. I. *Tab. XVI.*

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 6.

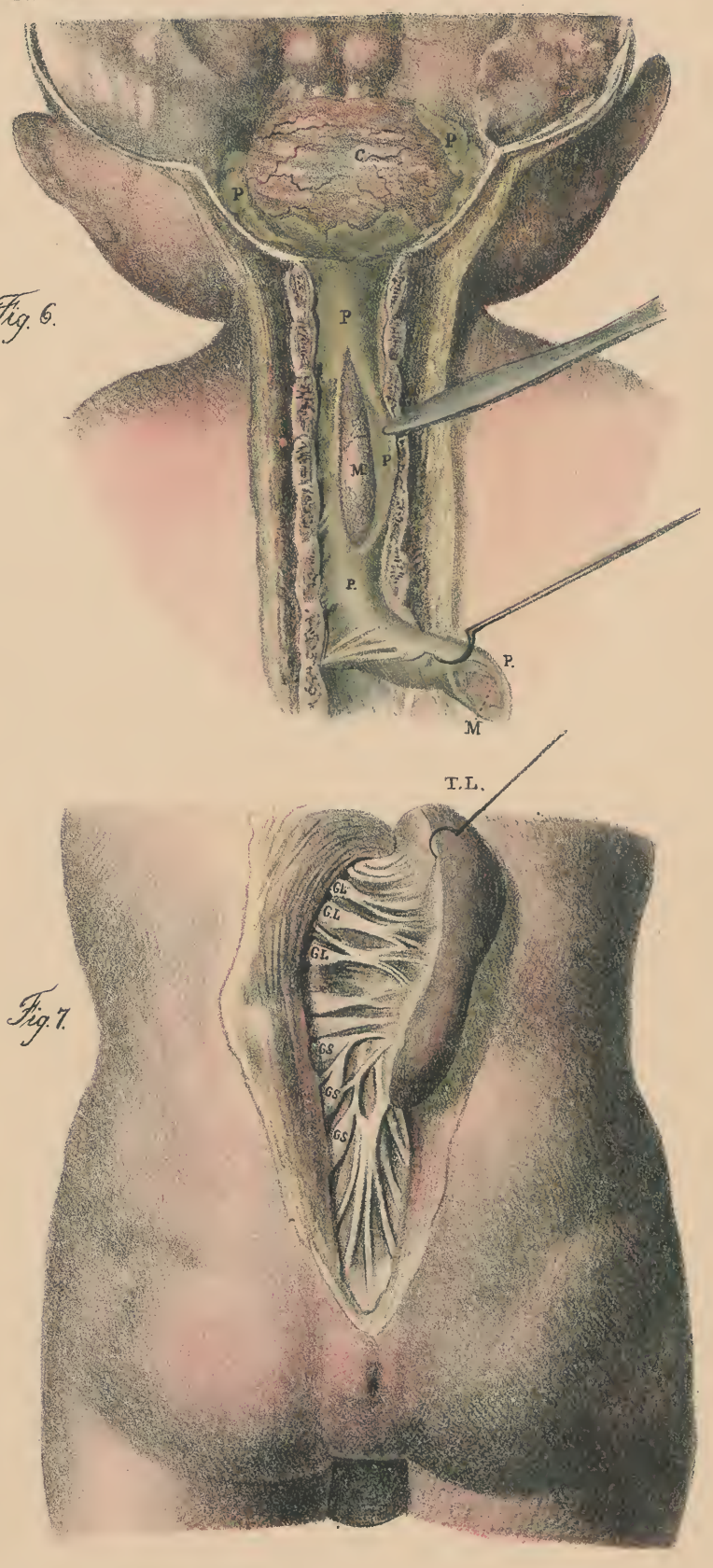


Fig. 7.

Fig. 4.

Fig. 5.

Fig. 8.

Fig. 9.

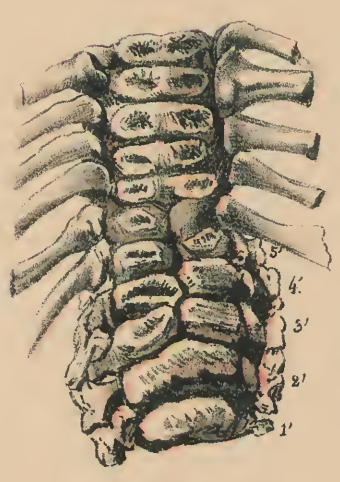
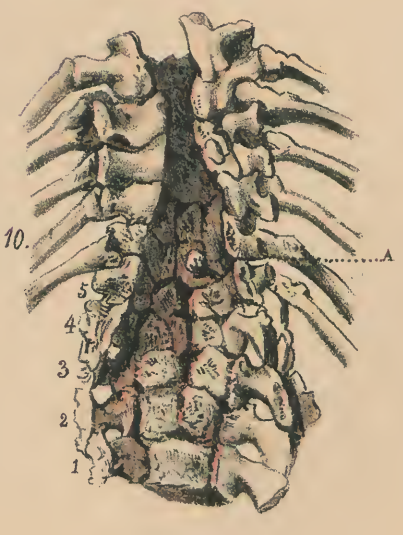


Fig. 10.



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Sec. II. *Tab. I.*

DISEASES OF THE HEART.

PERICARDITIS-

Fig. 1

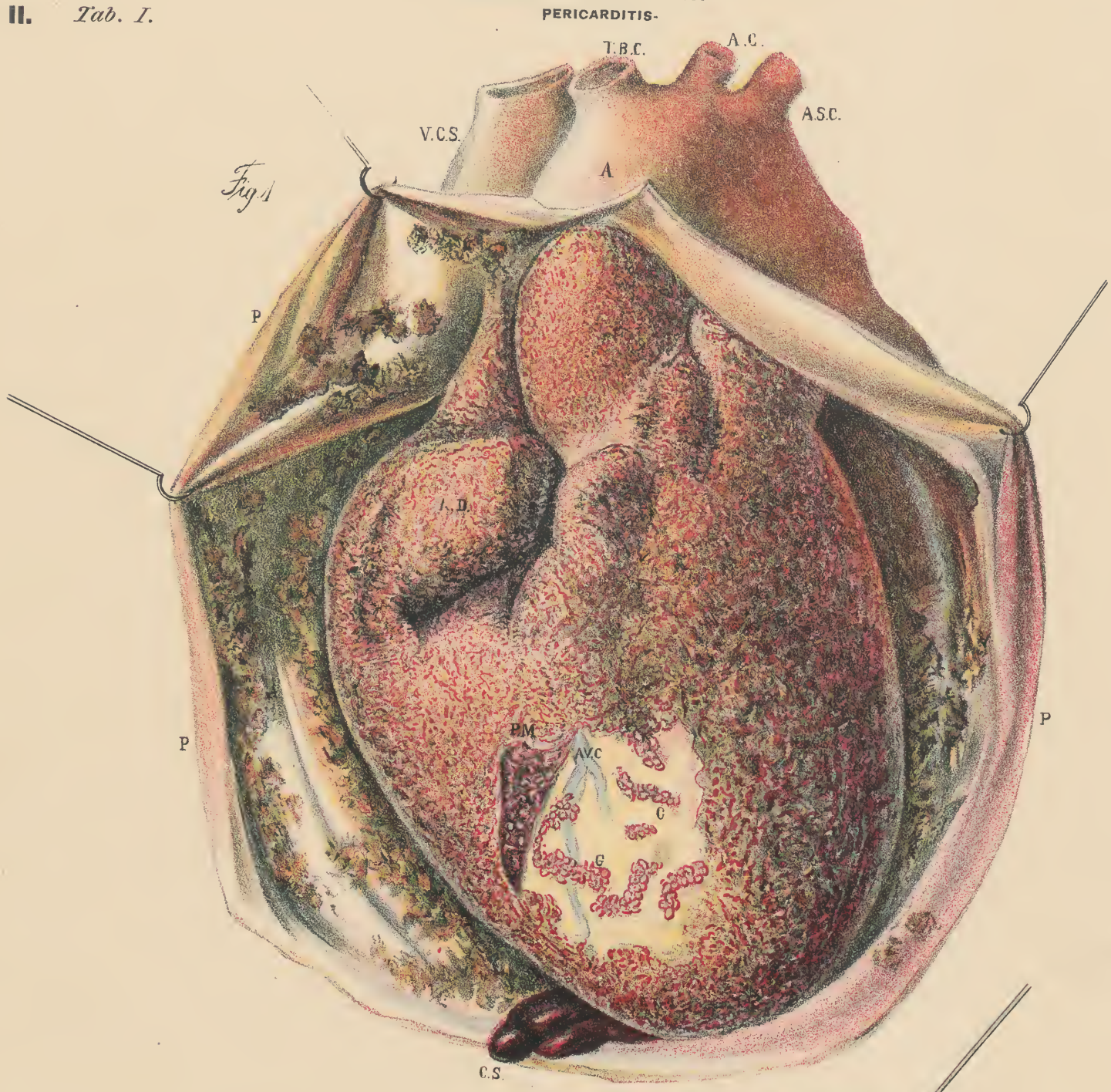
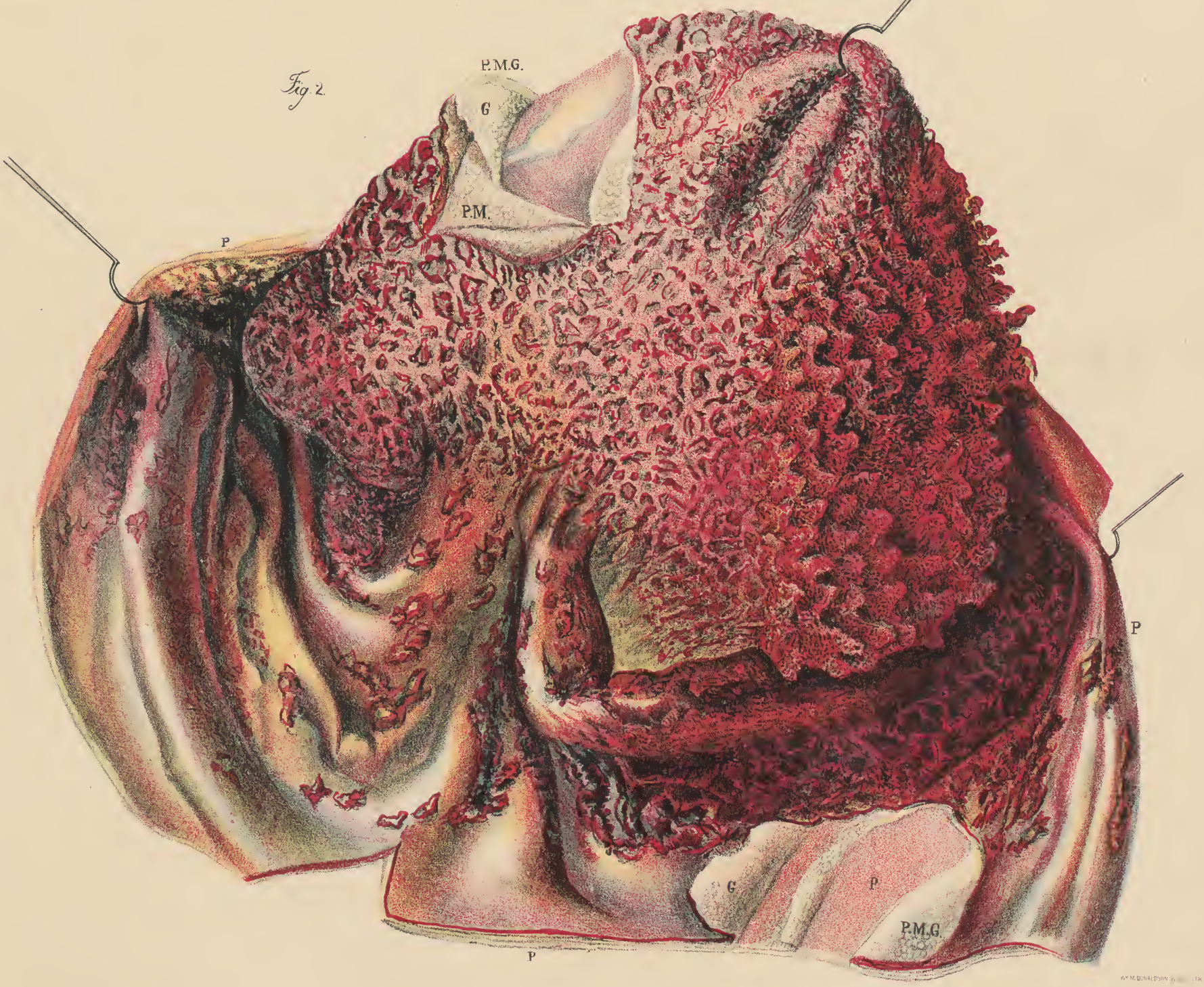


Fig. 2.



DISEASES OF THE ORGANS OF CIRCULATION.

DISEASES OF THE HEART AND ITS MEMBRANES.

Section II.—Table I.

Figures 1 and 2 represent two cases of Hæmorrhagic Pericarditis. Fig. 1 has no clinical history. Fig. 2 presented only dyspnœa and delirium as symptoms. An enlargement of the heart was diagnosed during life, but no pericarditis.

Autopsy. — Fig. 1. The heart is of a normal size. The pericardium is very much distended. A great quantity of sero-sanguinous liquid flowed out as soon as the sac was opened. The surface of the heart was covered with a pseudo-membrane of a deep brownish-red color; thick and wrinkled, very little coherent in its superficial layer, and very cohesive and granular in its deep layer, and attached to the visceral layer. The heart was neither changed in texture nor color. The subserous cellular tissue was neither infiltrated nor injected.

Fig. 2. The heart was very large; enlargement caused by distention of all its cavities. It is here turned up to show its posterior surface. The pericardium is enormously dilated. The whole surface of the heart was covered with a very deep red pseudo-membrane, very thick, areolated, and covered all over with conical projections, of different sizes and forms, which chiefly were situated on the left border of the heart. Altogether it had the appearance of a large *pine cone.*

A great number of long filaments, some thick, some very slender, projected from the heart. The pericardium was also covered, but with a smooth pseudo-membrane, on its inner surface. Some projections were situated here and there, and seemed to be continuations of those of the heart. The pseudo-membrane was formed of two distinct layers: (1) a superficial, clotty, very dark brownish-red, little cohesive and somewhat elastic; and (2) a deep, of white color, covered with tubercles or granules, very cohesive, easily separable from the superficial, but sufficiently adhering to the serous covering of the heart. On separating the latter, short strings which united them were seen; also red spots upon the whole parted surface. These were the newly-formed vascular net-works, covering the entire surface of the pseudo-membrane. The presence of two pseudo-membranous layers, of very different aspect, seems to indicate two distinct periods of the pericarditis. The granular layer, covered with vascular net-works, and very adherent to the walls of the heart, must be of prior date than the soft elastic layer, which seemed not yet organized. The clot granules of the serous membrane must not be confounded with tubercular formations. They are never converted into real tubercles, but become either eventually hardened or are gradually absorbed. Tubercular granulations, however, may readily be formed not only in old, but also in very recent, pseudo-membranes of the pericardium. As a general rule, they are associated with pulmonary tuberculosis and pleural exudations existing in chronic pneumonia. In such cases, the tubercles form, very often, dense, agglutinated masses, highly vascular, and very elastic, and cover the heart and pericardium.

The deep red color of the pseudo-membrane was, here, due to a quantity of bloody serum with which it was saturated. In cases where the pericardium is much distended by profuse exudation, the heart is pressed down deep in the pericardial cavity, and the exudation occupies its upper portion. It also presses the lungs back from the anterior thoracic wall. In this way the space of dull sound extends upward, and its limits may reach the third, second, and, in extreme cases, even the first intercostal space. The left sternal border, at first, constitutes its inner boundary, gradually it extends its limits downward and to the right, and but slightly to the left and outward. Thus, a very large triangular space of dull sound is formed in the anterior portion of the thorax. In voluminous exudations the heart-pulse is felt much below its ordinary position. It may then be felt either over several intercostal spaces, or may become perfectly imperceptible, especially if the patient occupies a supine position. When the patient stands up, the pulse will at once become perceptible, but the dull sound will then extend to the left far beyond the locality of the pulse. The cardiac sounds, when auscultated, will be very feeble, and frequent, and seem as if they came from a distance. There are cases in which the exudation compresses the basis of the left lung until it becomes solidified; under such conditions the hepatized portion of the lung will still further increase the area of the dull sound, and make it appear as if the exudation be more extensive than it really is. Auscultation over that region will then prevent an error of diagnosis. The most constant symptom of pericardial effusion is, an increase of the triangular space of dull sound in the cardiac region. As a rule, when the dullness on percussion extends much farther externally than the pulse of the heart, and the radial artery pulse is at the same time stronger than that of the heart, exudation within the pericardium may be predicated.

TABLE II.

Hydro-pericarditis.

Case. — Rup, a shoemaker, thirty-four years old; entered hospital May 23, 18—. Face of a purplish color, and bloated. Lower limbs much infiltrated.

Symptoms. — Breathing, dyspnœa and orthopnœa; oppression so great that he can neither inspire nor expire completely. Thin, wriggling pulse; frequent, irregular, about 110. Heart-pulse very feeble and almost inaudible to the ear. Application of the stethoscope fails to carry the sound. Dull sound over the region of the heart very extensive, nor is the heart-beat perceptible. Some effusion in the abdomen. The region of the liver somewhat sensitive to the touch. No signs of pleuritic effusion. Bronchial Rale, indicating induration of the apex of right lung. Was treated with digitalis. The next day, some relief of orthopnœa, but of only short duration. From the 8th of June, difficulty of breathing increases. Oedema of face, and some in the body. At very rare intervals he can lie on his right side, otherwise he maintains a sitting posture; the right side is, therefore, most infiltrated. Continued exploration of the thorax shows an increased dull-sounding space, but no infiltration in the pleural cavity. Continued bronchial respiration at the apex of the right lung, and a coarse mucous and sibilating Rale in other portions of that lung. Crepitus sound in the middle portion of it. Pulse still very irregular and feeble.

June 17. Has difficulty in swallowing, from excessive shortness of breath.

June 20. Hæmoptysis. Almost suffocating; can only sit up with his head very high and leaned on the right side. The following days, continues to spit blood. Abdomen very tender; orthopnœa; unable to move; excessive perspiration; almost pulseless. Died on the 28th of June in full possession of his senses.

Post-mortem Autopsy.—In opening the thorax, an incision was accidentally made in the pericardium, and about 500 grams of a brownish serum ran out and caused a partial collapse of the sac (Fig. 1). The pericardium was very much dilated. Fibrinous strings of different thickness (*A. A.*) extended from the heart to the pericardium. They were most numerous at the apex. The whole surface of the heart, the pericardiac portion covering the aorta and the pulmonary artery, were covered with a whitish, blistery pseudo-membrane, of little cohesion, which had a velvety appearance. On the posterior surface of the heart, thick pseudo-membranous cords running from the base to the apex. The pseudo-membrane lining the pericardium was much thinner than the cardiac.

Fig. 2 represents a section of the heart, showing the thickness of the pseudo-membrane. Its deeper layer, apparently of older date, was far more dense and firm than the newer or superficial, which was loose and friable. The pericardial pseudo-membrane was of the same character. The lungs were compressed and pushed backward, and only of half their ordinary size, for the pericardium occupied the greater part of the anterior thoracic cavity. The base of the left lung and the corresponding pleura were also invested by a similar exudation as the pericardium, which seemed of recent formation (*F. I. P.*—Fig. 1). The basis of that lung presented on its circumference traces of superficial pulmonic apoplexy. The right was so adherent to the costal pleura, especially at the apex, which was indurated and bore traces of old tuberculous deposits, that it could not be separated from it without tearing. This whole lung was œdematous, congested, and barely permeable to air.

The absence of protrusion of the thorax over the distended space, notwithstanding the considerable quantity of effusion within the pericardium, is remarkable in this case. There is a great difference in different individuals in regard to that. On some a very distinct ridge is formed over the costal cartilages and left rib, corresponding with the region of the heart. In others the enlargement of the heart and pericardial distention is formed at the expense of the lung, which it compresses, and whose space it will occupy, and therefore no protrusion anteriorly will be formed.

In fact, a clear distinction must be made between an aneurism of the heart with an hypertrophy and enlarged organ and an aneurism with attenuation of its walls; between diseases of the heart proper and exudation within the pericardium. The greatest protrusion of that region of the thorax was mostly observed in cases of dilatation with hypertrophy of the heart. In pericardial distentions alone, the liquid presses upon all sides with an equal force and depresses the diaphragm (which is the most yielding) very much, and hence the protrusion forward is thereby much diminished.

Symptoms and Pathology.—In relation to the *ætiology* of pericarditis, it is necessary to state, that *idiopathic* pericarditis is of very rare occurrence. It is, as a rule, a lesion associated with many varieties of inflammations of other serous membranes, such as the pleura, the peritoneum, etc., and the synovial structures. It is a most common complication of inflammatory rheumatism. Bouillod, Cruveillhier, and Hunter have sufficiently indicated the ætiological relation of rheumatismal synovitis to pericarditis. Diseases of the lungs of the graver forms, which are always complicated with inflammation of the pulmonary pleura, are, not seldom, the primary cause of pericarditis. Dieulafoy, in his report of two cases of pericarditis, complicated with purulent pleuritis, remarks, that in acute pericarditis exudations are very seldom found; in twenty-two cases which he observed, in only four was there exudation in quantity sufficient to clinically diagnose. Two of these were complicated with tubercular, one with purulent pleuritis, and one with acute rheumatism (*Gazette Hebdomad, No. 29*). Riess calls attention to a symptom, which has seldom been noticed, indicating attachment of the pericardium or synechia of both plates; that is, a metallic ring of the sounds of the heart, following the cardiac rhythm, audible at a distance of a yard or more from the patient. When the pericardium is immovably attached to the diaphragm, the same sound exists, but is not so loud as the former (*Berlin Klinish, Wochenschrift, No. 51*). Widman considers the drawing in of the intercostal space, during the systole of the heart, a valuable symptom of attachment of the pericardium (*Virchow's Archiv, Vol. LXX.*). Riegel and Tuczeck call attention to the fact that the so-called extra-pericardial attachments manifest themselves by a weakening of the heart-pulse during expiration. Certain cordiform attachments between the lung and pericardium become relaxed in inspiration and tense during expiration. The heart does, therefore, become free in its motion during inspiration, whilst during expiration the pericardium is retracted with the receding lung, and the heart-pulse is therefore reduced in force (*Berlin Klinish, Bericht, No. 29*). Francois Frank (*Gazette Hebdomad, No. 29*) states that a pericardial pressure (as produced by pericardiac effusion) causes enormous reduction of the arterial pressure, whilst the heart continues its rhythmic action unchanged.

TABLE III.

Tubercular Pericarditis with degeneracy and tubercular infiltration of the right auricle.

No clinical history of patient. The pathological specimen represented here sufficiently explains the nature of the lesion. The right ventricle (*V. D. V. D.*) is laid open and spread out. The pericardium is dissected away from the left ventricle, to which it firmly adhered. The connective tissue which united the two plates was exceedingly dense, and was filled with tubercular masses (*M. T.*). In the auricle the tubercles were more abundant than in the ventricle. The pericardium was completely blended with the auricle, the aorta, the pulmonary artery and the superior vena cava. Not only was the fibrous tissue of the auricle perfectly filled with tubercles, but the muscular tissue of the auricle was completely obliterated by the large tubercular masses. Only the endocardium was not affected by it. The superior vena cava was almost altogether changed by the same mass, whilst around the aorta and the pulmonary artery it formed a perfect cylinder, without, however, injuring the arterial tissue proper. The circulation within these arteries must have been much impeded. The contraction of the right auricle must have become impossible. (This fact may lead to the conclusion, that contractions of the auricles are not absolutely indispensable to carry on circulation.) Breitung found, in 419 cases of pericarditis, only two cases of idiopathic tuberculosis of the pericardium, and only *one* case of tubercles in the pericardium alone, and nowhere else. Associated with other tubercular affections, and as complications, he found about forty cases (*Berlin Dissert*). Biron (*Thêse de Paris, No. 4*) states that this rare idiopathic form is found mostly in early childhood. He differentiates two forms of that lesion. One without *any exudation*, but always producing adhesion of the two plates or of synechia with the whole heart; and one with profuse exudation. The first form is impossible to be diagnosed unless it be associated with a tubercular diathesis, when the general cachexia might lead to surmise such a lesion. The second form is the exudative, but does not differ in its symptoms from ordinary effusions within the pericardium.

FIG. 2. *Acute Hæmorrhagic Myocarditis with Apoplectic Focuses.*

CASE.—A woman, about sixty years old; showed symptoms of dilatation and hypertrophy of the heart for some years. Died suddenly. A post-mortem examination showed the heart covered all over with clotted blood, which filled up the whole cavity of the pericardium.

DISEASES OF THE HEART.

PERICARDITIS-

Fig. 1.

P
P
A
A
A.P.
A.P.
A.S.C.
A.C.
T.B.C.
P
P
P
F. I. P.

Fig. 2.

P.M.
P.M.
C.V.S.
C.V.D.

Sec. II. *Tab. III.*

DISEASES OF THE HEART.
TUBERCULAR PERICARDITIS.
HAEMORRHAGIC MYOCARDITIS.

Fig. 1.

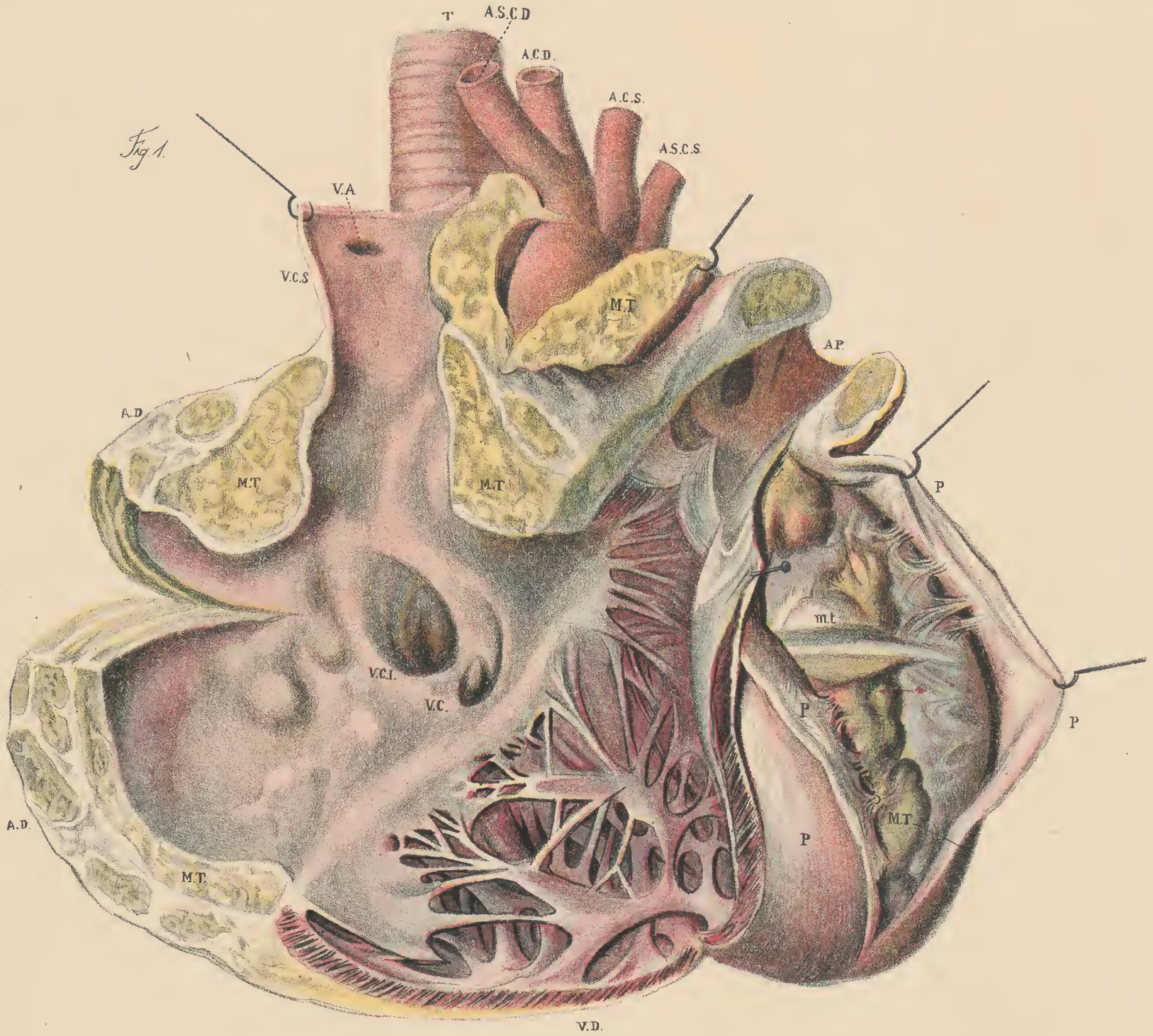
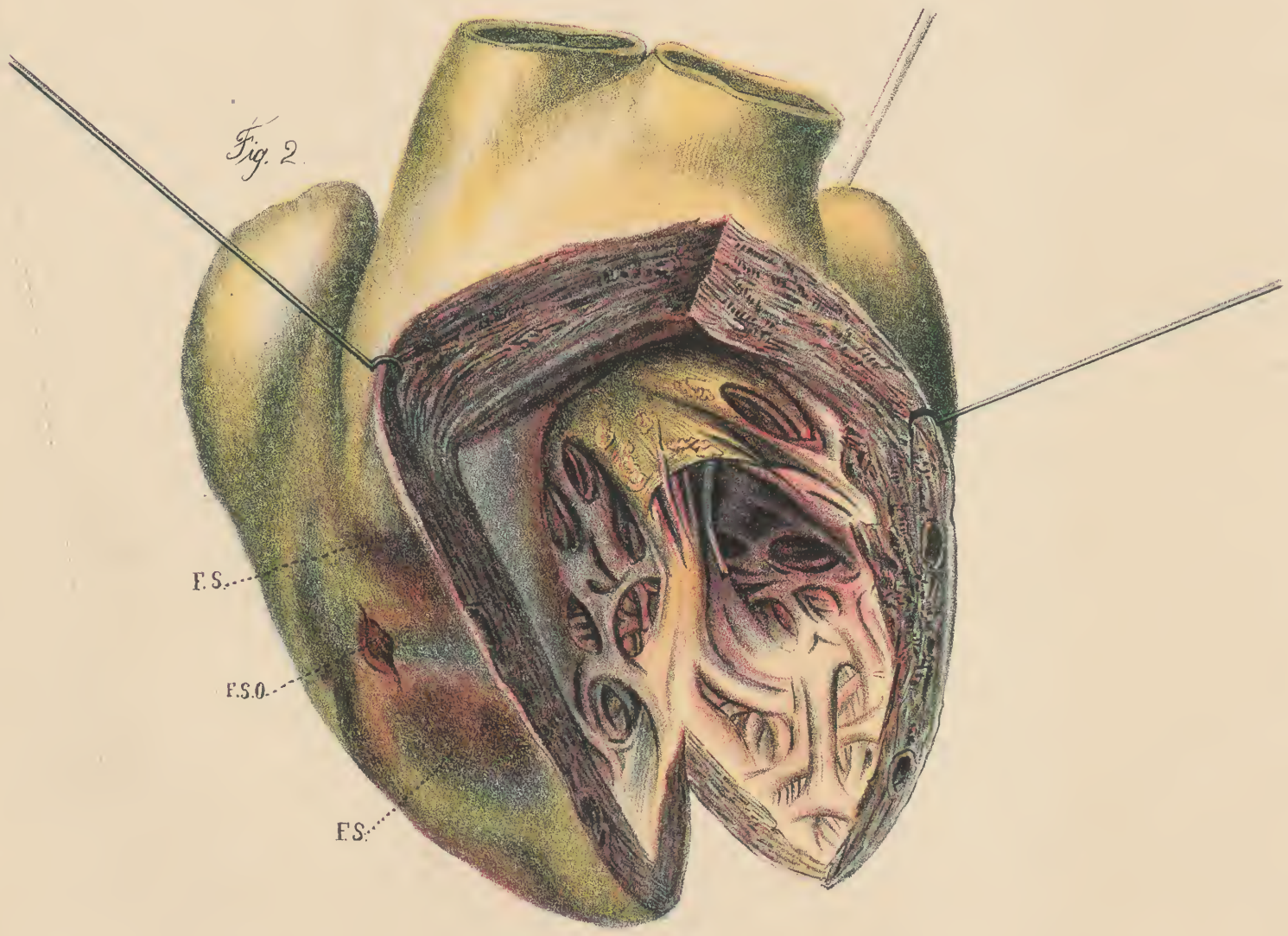


Fig. 2.



Some liquid blood was also found in this cavity. On the left ventricle some blackish ecchymotic spots proved to be apoplectic focuses (*F. S. F. S.*—Fig. 2), situated immediately below the visceral plate of the pericardium, which they had raised (*F.*); it showed an opening or rent (*F. S. O.*), which seemed to be the source of the pericardiac hæmorrhage, and must have come from the left ventricle. The left ventricle was both hypertrophied and dilated. The left auriculo-ventricular orifice was narrowed. The aortic orifice exceedingly narrow, and obstructed with very large irregular phosphatic concretions. In this case, the muscular tissue of the heart had undergone, in certain localities, a gradual softening and disintegration, and at last broke down, in the same way as cerebral apoplexy is brought about, by gradual disintegration of some of its tissue, with infarctus and focal hæmorrhage. This breaking down, which does often take place through the whole thickness of the walls of the heart, and which causes sudden death, happened here only to a portion of its thickness; more at the outside than the inside of its walls; the hæmorrhage took place outside and the blood discharged into the pericardial cavity. Had an extensive focus formed on the inner part of the wall, and broken internally, the blood of the ventricular cavity would have penetrated into the little cavity of the infarct, and the heart would have sooner broken through. It might possibly sometimes happen, that the walls of the heart resist the influx of blood from the ventricle, and a diverticular cavity, similiar to a false aneurism, would be the result. The constant forward movement of the blood, coming from the ventricular cavity would so irritate that portion of the heart, that it might cause a repair either by inflammation or by cicatrization; or it might undergo a fibrous, cartilagenous or osseous transformation. Such transformation of the walls or parts of a diverticulum, or pocket, in the heart-walls are not so very rare; but they differ totally from the mechanism of an aneurism, either of the heart or of an artery. Cases of this kind may be classed as chronic diffuse myocarditis. The symptoms accompanying such lesions are various. On account of the inability of the ventricular walls to fully perform their function, there will generally be found an insufficient blood pressure in the aorta, with consequent venous engorgement; oedema, as well as passive hæmorrhage, will appear and disappear alternately in different organs, as constant and early symptoms of the disturbance of the circulation of the heart and lungs; there will always exist shortness of breath, moderate dyspnœa, a sense of suffocation after a strong effort, and palpitation, with some pain in the region of the heart. In nearly all such cases there is a disturbance of digestion. The arterial pulse is nearly always irregular. The same irregularity is found in the, otherwise, clear heart sounds. In some rare cases there is a slight systolic Bruit heard at the apex. In the early stages a fairly distinct heart-pulse may be discovered by palpation, but of rather irregular strength. In the latter stages, no pulse whatever, even at the apex, can be discovered. Percussion will indicate an enlarged space of dull sound, especially to the left. Whether an arterial and cardiac irregular pulse may not also indicate an endocarditis, or fatty degeneration of the muscular walls of the heart, or valvular troubles, is not yet decided; for in many cases of valvular diseases and *stenosis* of the left ostium venosum, great irregularity of both the cardiac and arterial pulse is often enough observed. But under such circumstances, the irregularity of both pulses may still be due more to myocarditis complicated with those diseases, than otherwise. A systolic Bruit, when only occasionally heard, or when a tone is heard simultaneously with it, does not indicate an insufficiency of the mitral valve in myocarditis. The ætiology of diffuse myocarditis seems chiefly rheumatic affections, especially of the muscles, with over-fatigue of the heart. Puerperal and miasmatic myocarditis do not differ in their symptoms from other forms of this lesion.

TABLE IV.

Spontaneous Rupture of the Heart.

The heart represented in this plate (Fig. 1, 2) was that of an old woman, eighty-six years old. She died suddenly.

Post-mortem Autopsy. — After the sternum was removed, the pericardium was found considerably distended, and of a purplish-blue color. It contained some liquid blood, which ran out on opening the cavity, and some coagulated blood, which enclosed the heart, and extended to the pulmonary artery.

Fig. 1 presents two perforations (*P. P.*) on a plane with the ventricular septum beneath the pericardium. Several hæmorrhagic spots and ecchymoses are to be seen in the space between the perforations.

Fig. 2 presents the left ventricle opened by a vertical incision made along the track of the perforations. The inferior is the largest, and is situated near the ventricular septum, and consists of a rent in the tissue of the heart; the infiltration took place from below upward, between the serous membrane, which is covered with fat and the *columnæ carneæ*, and extended — much farther than the superior perforation (*P.*—Fig. 1) indicates — behind the pulmonary artery, between this and the aorta, where a hæmorrhagic focus had formed. There was no general disease of the parenchyma of the heart visible to the naked eye. Neither was there any stenosis of the orifices, nor hypertrophy or alteration of the tissues generally, except a spot near the perforated portion, which was very friable, and which was proven by the microscope to be a portion of muscular tissue, softened by amyloid and partly fatty degeneracy.

Fig. 3. *Pseudo-membranous Pericarditis in a three-days-old babe.*

The heart is enveloped by a neoplastic layer (*F. M.*). The pericardium is covered with a similar layer, but thinner than that of the heart. This pericarditis seems to date from a time before it was born. Newly-born children are often affected with all sorts of inflammations of the serous membranes.

Pathology. — Condition of the pericardium in rupture of the heart. — In most of the cases of rupture, especially when the rent is very large, the pericardial cavity is distended, and filled with the extravasated blood. When the orifice is small, especially if the tissue which broke down and suffered the rupture has not been extensively degenerated by fatty or other transformations, such as amyloid, etc., of the muscular fibres, cicatricial tissue is often speedily formed and repair allowed, provided the clot between the heart and the pericardium remain for a time stationary. Such ruptures, if closed up, cause attachment of the visceral plate (and nearly always the outer plate also) to the heart by fibrinous strings or bands. During life, such slight ruptures are barely discoverable. When the internal surface of the infarctus heals, there is produced either a slight contraction of the cicatrized portion, or some callus projecting into the cavity of the heart, which, if not of any considerable size, is also not noticeable during life.

As a rule, an extensive rupture causes sudden death; for, not only will the extravasated blood within the pericardium prevent diastole of the heart, but the vessels enclosed within the membrane will be completely compressed, and sudden cessation of the circulation will ensue. Besides, in spontaneous rupture, the muscular tissue of the heart must have undergone such a change, and have become so weakened as to be unable to resist the force of the passage of the blood through the cavities. Outside of traumatic causes, endocardial ulceration, most frequently existing in infectious diseases, and puerperal metritis, septicæmia, and the graver forms of pleuro-pneumonia, most commonly give rise to rupture.

General Pathology. — Rindfleisch in his Pathology very correctly remarks, that myocarditis, or inflammation of

the parenchyme of the heart, is the most obscure chapter of the whole pathology of the heart, and, unfortunately, pathological anatomy can throw but a feeble light on the subject. Experience has taught us that the voluntary muscles of the body, even in the slightest state of inflammation (though there be no hyperæmia present), produces very grave functional disturbances in that tissue. The muscle rests in a state of contraction, and the slightest effort at extension meets with the greatest resistance on the part of the patient, on account of the great suffering it produces.

Judging from analogy, it is easily conceivable that even the lowest grade of a general inflammation of the myocardium would bring the heart to a stand-still, and produce sudden death; that only in partial affections could the latter stages of inflammation become possible. Theoretically, diffuse myocarditis is impossible, yet not only a large portion of the muscles of the heart, but also the whole heart, may, under certain circumstances, undergo the inflammatory process. Of course, death will always be the result. The following case will show it: A man, fifty years old, who was treated a long time for constitutional syphilis, and who was lately attacked with inflammation of both lungs, died suddenly. It was supposed that the sudden death was due to cerebral apoplexy, but a post-mortem examination revealed that, with the exception of some insignificant syphilitic productions, there was a condition of the heart which might well be considered as a general parenchymatous inflammation of its muscular tissue. The organ was contracted, and its walls so rigid that it required considerable force to compress it. Even after it was opened it did not collapse. The muscular tissue had lost its fresh red color, and was of a violet tinge. The cut surface of a section had a glistening appearance, and its edges were almost transparent, and of the consistency of India rubber. The fibres could readily be torn, but not stretched. Beneath the pericardium, as well as in the endocardium, were very numerous ecchymoses, which were probably caused by the grave disturbance of circulation in the myocardium (*acute anæmia*). All the vessels were empty, and the muscular tissue must have been in the highest degree anæmic. Microscopic examination showed a finely granular appearance of the interior of the muscular fibres. The granules were mostly distributed in the vicinity of the nuclei. The fibres were torn into short fragments; a phenomena constant in pathological conditions of the striated muscles. It represented the most perfect type of Virchow's parenchymatous inflammation.

Hypertrophy of the Heart.

Cardiac hypertrophy is an increase of volume of the heart, caused by hyperplasia of the myocardium. It may befall either ventricle, or both at the same time. The former is most frequently the case. The hypertrophic condition of a ventricle is not only an increased quantity of its muscular tissue, which is manifested by an unusual thickness of the ventricular wall, and a more or less changed form, due to an augmented volume, but it becomes harder and more rigid. When such a hypertrophied ventricle is cut open and emptied of its blood, its walls will not collapse, nor can they readily be bent, either in or out. The increase of volume of the heart, in hypertrophy, is not only due to increased substance, but also to dilatation of its cavities, and the change of form of the organ will be characterized by a peculiarity proper to each ventricle. In hypertrophy of the left ventricle, the heart will assume either an oblong-ovoid, or a cylindrical shape; the right ventricle will then appear as a mere appendix to the left. The position of the heart in the thorax will be almost horizontal (its base directed to the right, and its apex to the left), and will extend in that direction beyond the usual mammary line. It will be marked by displacement of the apex beat, which will be more to the left, and the extension of the dull cardiac sound farther in that direction. In right ventricular hypertrophy, there will be not only an elongation of the heart, but also a widening and a thickening of the whole organ. Seen in front, it will have an almost quadratic form. As it will tend to place the heart in a more vertical direction, it will be marked by an extension of the dull cardiac sound more to the right, and will reach the lower right border of the sternum, and even beyond it. In such a state, the apex will not only be formed conjointly by both ventricles, but in some instances the right ventricle alone will constitute it, and, as a consequence, instead of an apex beat (which will be mostly quite imperceptible), there will be a *basal* shock, because the thickened and enlarged base will bring in immediate contact, the *arterial cone* with the inner wall of the heart, and of course the shock will be perceptible at the base during the systole. Each cardiac hypertrophy is produced by overworking of the heart, in its effort to overcome mechanical impediments of circulation. The impediments increase the labor to be performed by the heart, by augmenting the pressure, which is directed perpendicularly towards the inner wall of the ventricle during a systole, and which pressure is itself to be overcome by the contraction of the ventricle. In atheroma of the aorta, there is always formed a hypertrophy of the left ventricle. In valvular disease will be found the chief causes of such hyperplasia; though difficulties of circulation, in other organs, do not a little contribute towards forming such cardiac conditions. The histological changes of the muscular tissue, in hypertrophy, is ascribed to increase in size and density of each muscular fibre. Such enlargement of the fibres seems due to increase of cellular elements in the fibre by *cell division*. It is well known that the muscular fibres of the heart bifurcate, and thus form net-works by the union of opposite bifurcations, leaving between them longitudinal fissures. These net-works can be reduced to small fragments with straight ends, which represent the cellular elements. Each of these have a central nucleus. In a hyperplastic muscle of the heart, there are formed cells, with many neuclei, arranged in rows, one behind the other.

Atrophy of the Myocardium.

In a muscular organ, like the heart, whose activity surpasses that of any other muscle of the body, a lack of nutrition is very soon and very sensibly felt. Not only does the heart speedily suffer from atrophy in old age, when the process of nutrition naturally becomes lessened, but also each cachexia, each anæmia, be it due to an acute or a chronic state of disease, can become a cause of cardiac atrophy, which will be characterized by thinness and flabbiness of its muscular tissue. This would constitute general atrophy. There exists atrophic conditions of only one layer of its muscle, or even of some localities or portion of the heart. These are partial atrophies. The latter are due to local anæmia. In all those cases the muscular fibres become thinner, more slender, or partly obliterated. The histological character of atrophy offers many modifications. They are as follows: Brown atrophy, as the name indicates, is manifested by an increase of volume, and at the same time a discoloration of the tissue, and turning into a brown or brownish-yellow color. This is due to the filling up of the contractile portion of the muscular tissue, with yellow pigment granules, arranged in a variety of ways between the primitive fibres and around the nucleus. Brown atrophy is an affection of the whole heart. It is found in senile marasmus, and in inanition, in tubercular and carcinomatous cachexia.

Yellow Atrophy or Fatty Degeneration of the muscles of the heart.

In a measure, as the muscular tissue undergoes the fatty change, it becomes more discolored; turns at first yellow, then gradually whitish-yellow, and has the appearance of tallow. It loses its consistency, and becomes friable; can easily be reduced to a pulp between the fingers, and increases but little in volume.

DISEASES OF THE HEART.

PERFORATION.

PERICARDITIS OF AN INFANT.

Fig. 3.

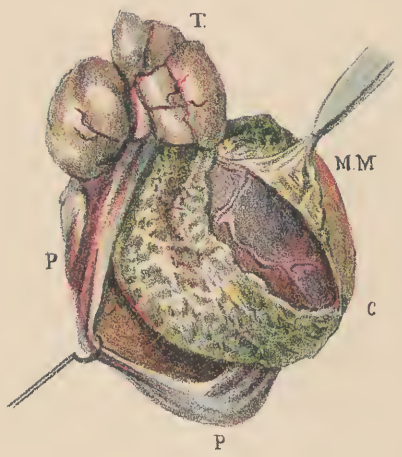


Fig. 1.

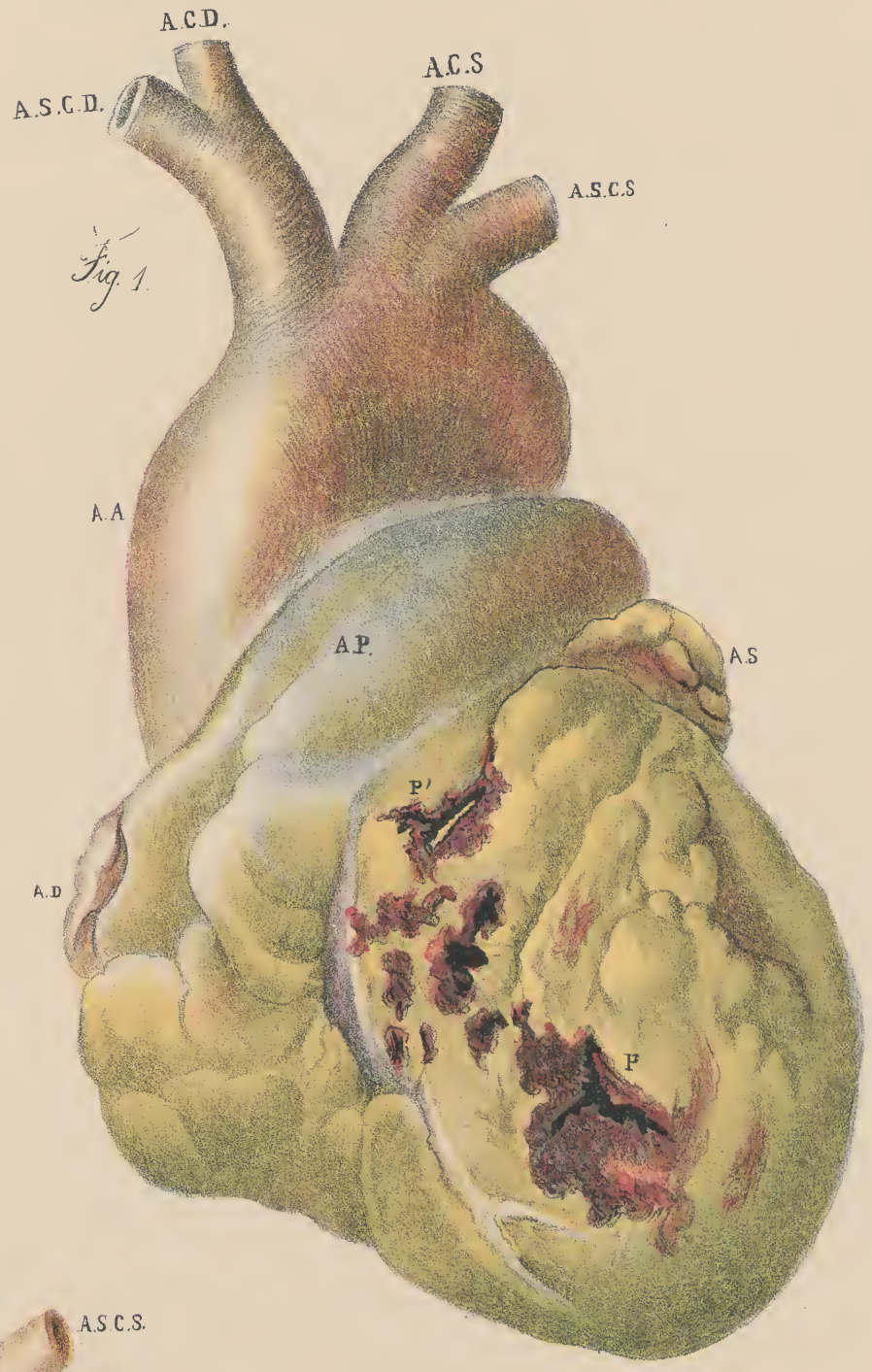
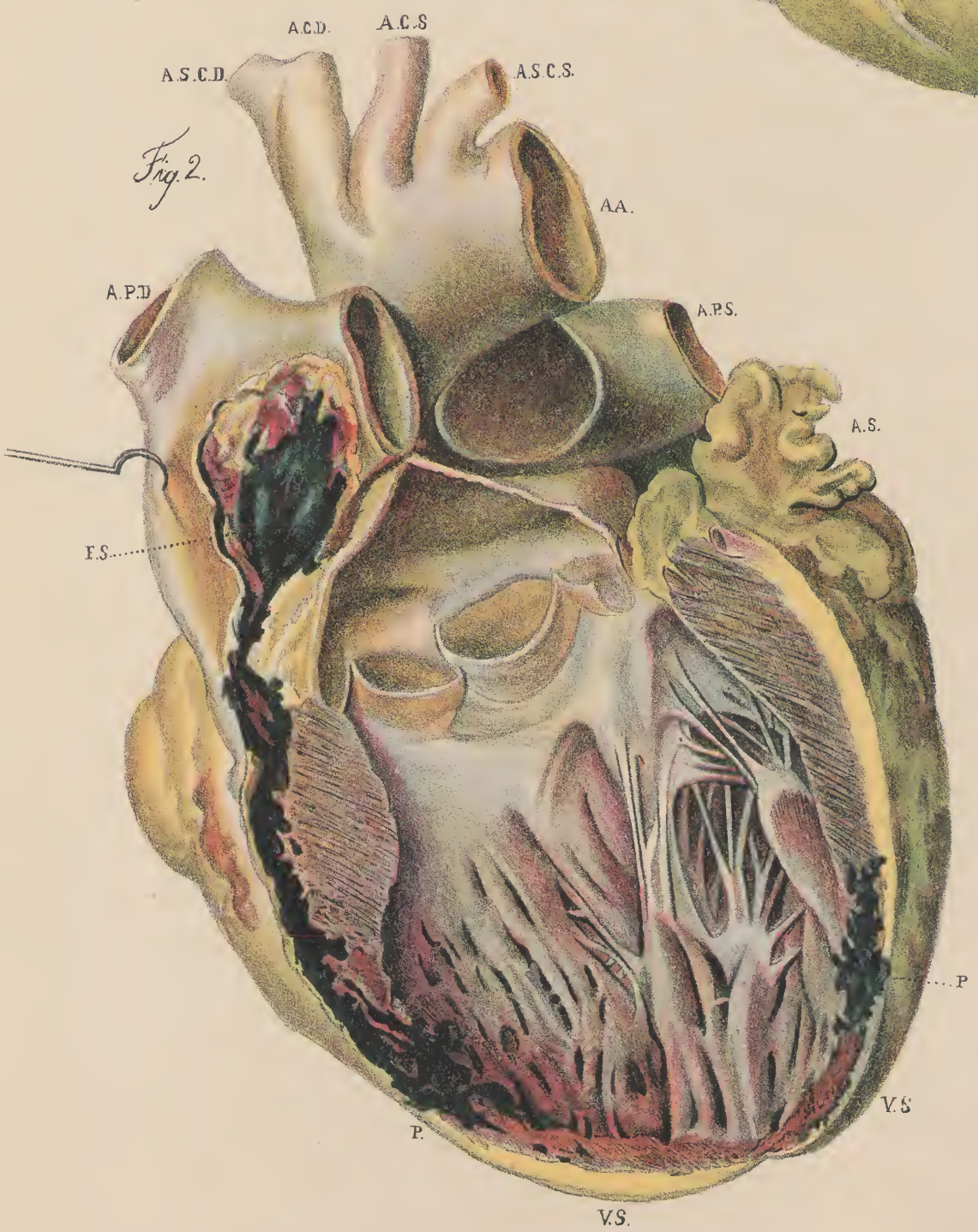


Fig. 2.



BROWN ATROPHY.

HYPERTROPHY OF RIGHT VENTRICLE.

Sec. II. *Tab. V.*

Myomalacia and Rupture of the Heart.

Fig. 1.

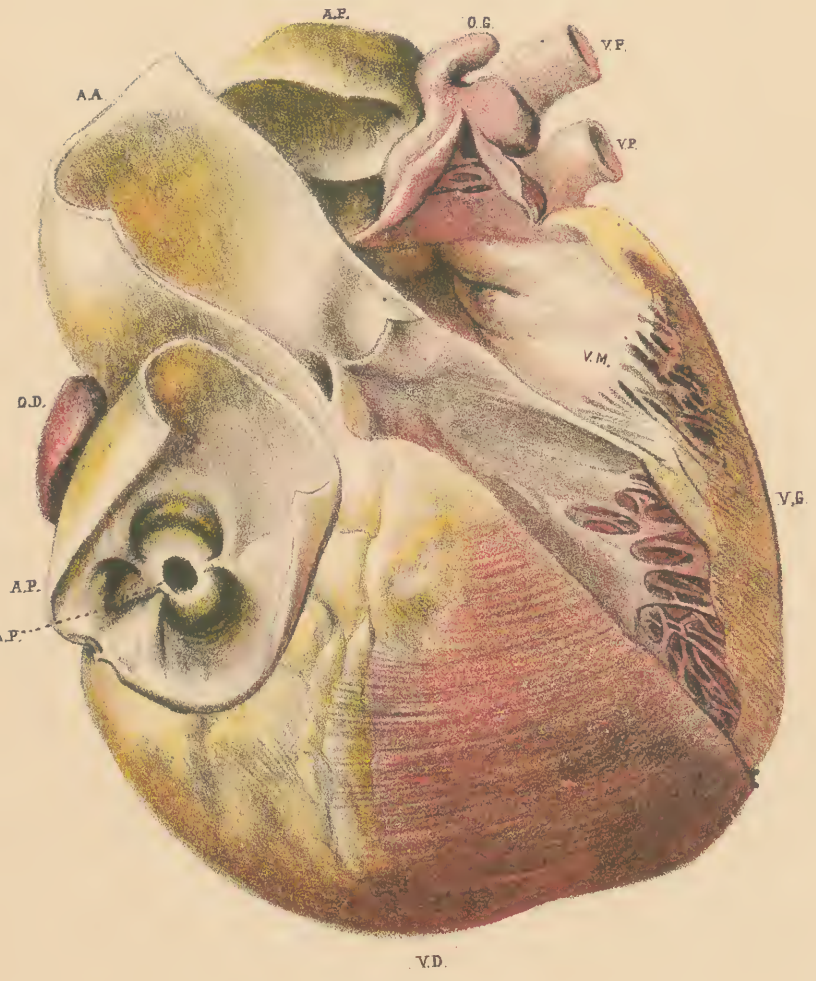


Fig. 2.

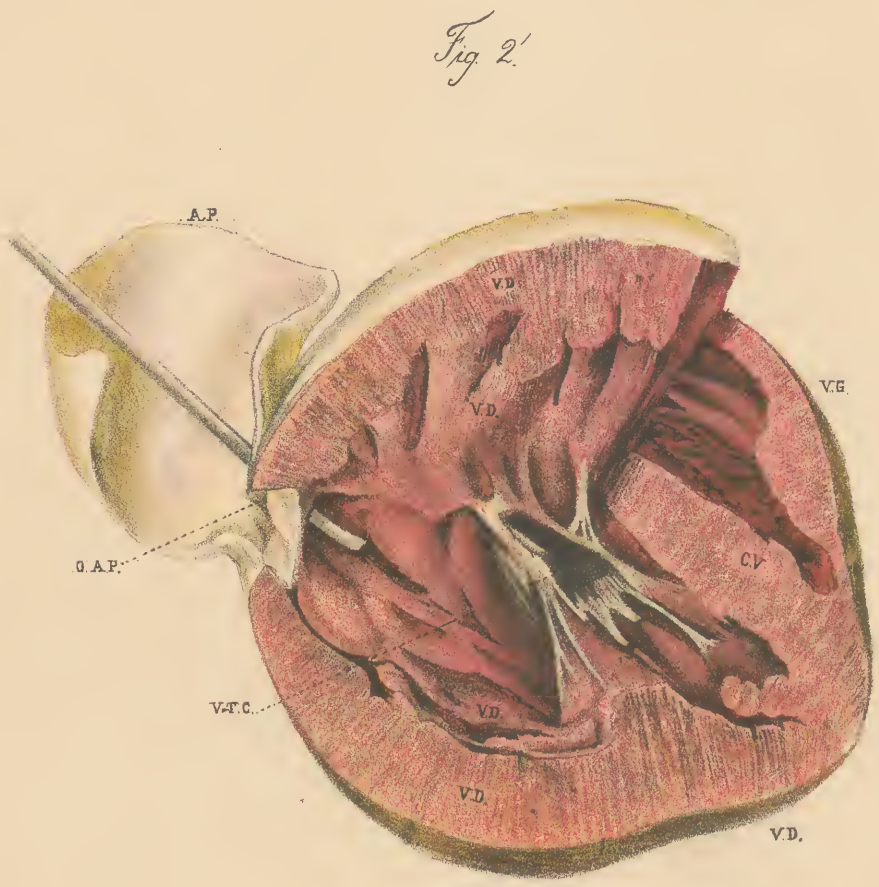


Fig. 3.

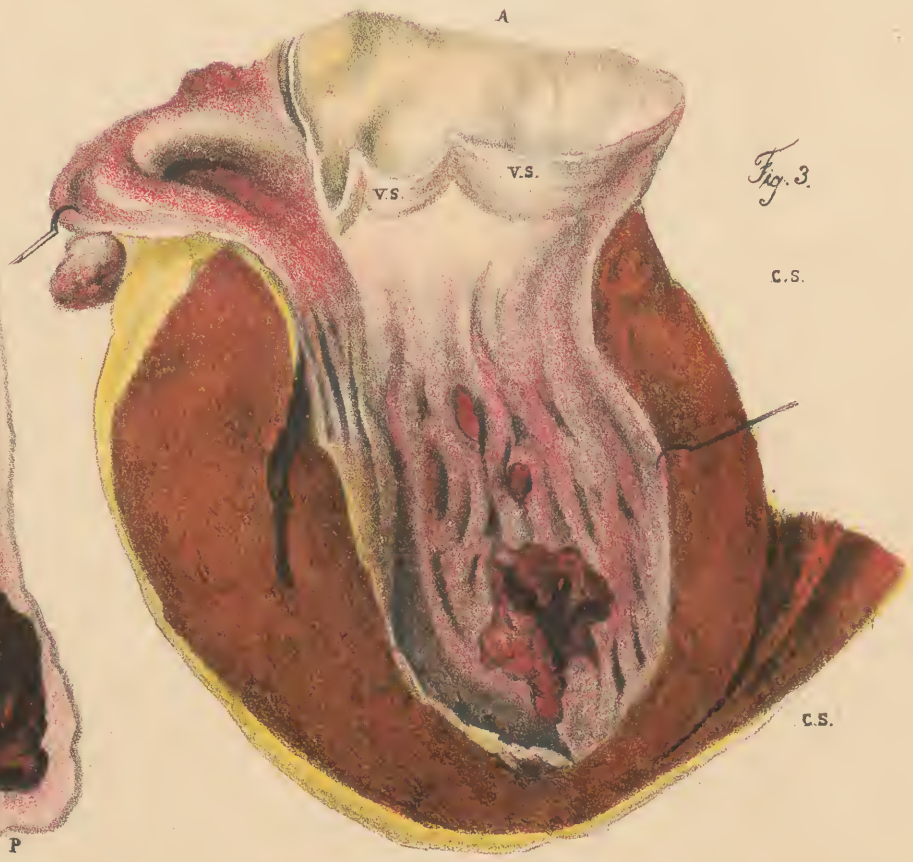


Fig. 4.

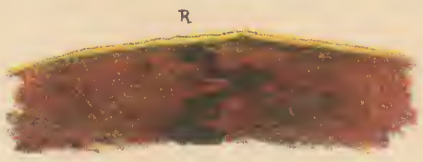


Fig. 5.

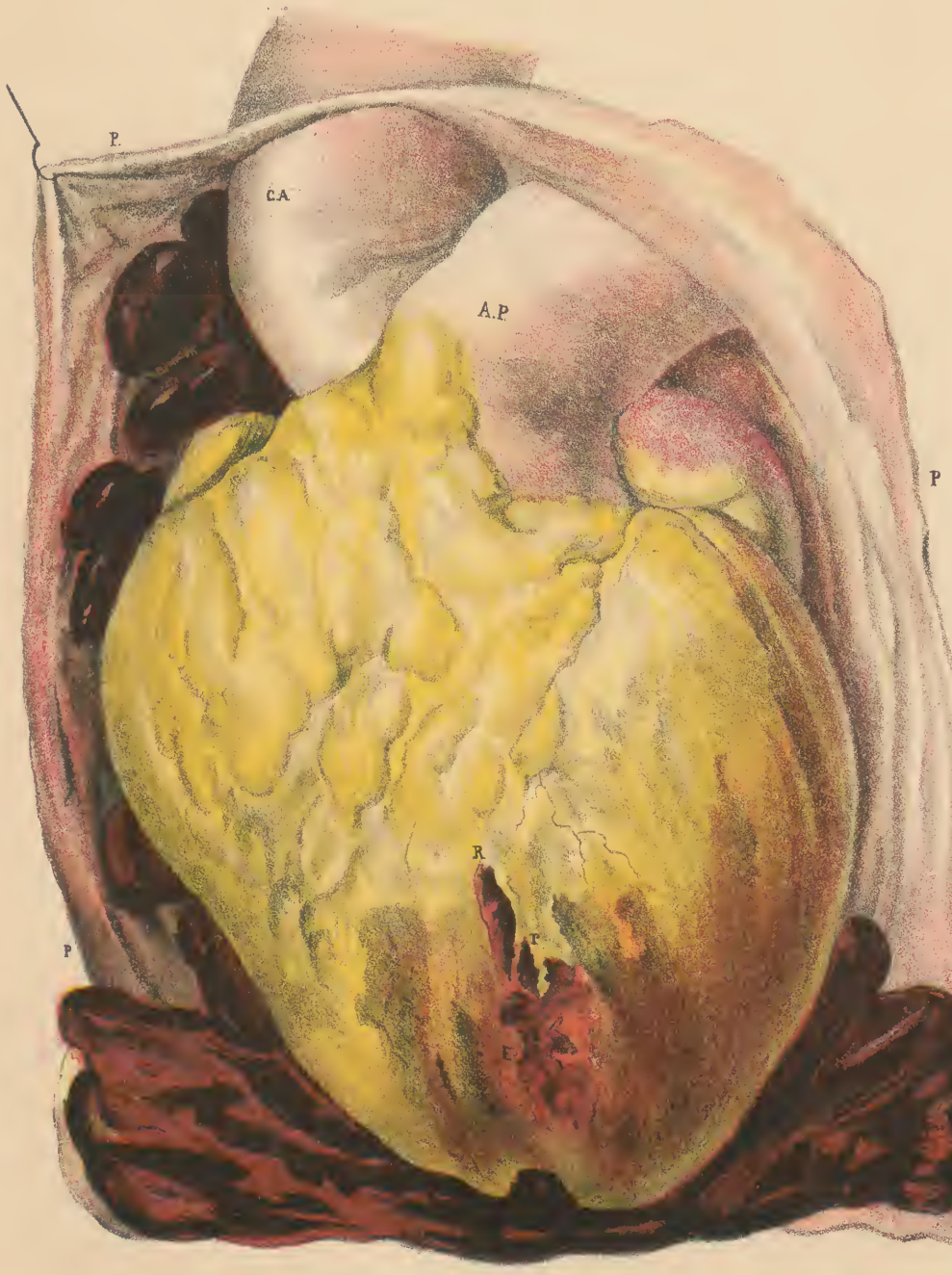


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TABLE V.

FIG. 1.—*Brown atrophy of the heart. Compensatory Hypertrophy of the right Ventricle. Stenosis of orifice of the pulmonary artery; complete atrophy of the semi-lunar valves. Excessive constriction of the right auriculo-ventricular orifice, with insufficiency of the tricuspid valve; Atrophy of the left Ventricle, atresia of its cavity, incompetency of the mitral valve, excessive atrophy of the left auricle.*

There is no clinical history of the case, only a few remarks in regard to it. Symptoms reported as follows: Bellows murmur, in consequence of friction of the blood, in passing through a very narrow orifice. General serous infiltration, from obstacle formed in the way of the venous current. Suffocatory attacks from incomplete aeration of the blood in the lungs. Cyanosis of the face and upper extremities.

FIG. 2.—*Longitudinal section of the heart through both ventricles.*—(*A. P.*, pulmonary artery; *O. A. P.*, ostium of pulmonary artery; *V. D.*, right ventricle; *V. S.*, left ventricle; *C. V.*, septum ventriculi; *V. T. C.*, tricuspid valve; *V. M.*, mitral valve; *A. D.*, right auricle; *A. A.*, arch of aorta; *A. G.*, left auricle; *V. P.*, pulmonary vein.)

FIG. 3.—*Section of the left Ventricle in a case of Myomalacia.* (*A.*, Aorta; *C. S.*, left heart; *V. S.*, semi-lunar valves.)

FIG. 4. *Ruptured portion of heart.* (*R.*, rupture.)

FIG. 5.—*Myomalacia and rupture of the heart.*–(*P.*, pericardium; *C. A.* arch of aorta; *A. P.*, pulmonary artery.)

Yellow atrophy differs from the brown variety in being, as a general thing, of an acute character and constituting a secondary or later stage of a number of highly acute diseases. It may form: 1, the sequel of acute, febrile diseases, such as high graded remittents, acute exanthemata, typhus and severer forms of typhoid; the cardiac lesion will be in the form of a degeneration of the whole myocardium; 2, a fatty degeneracy of the superficial subpericardial layer of the heart consequent upon nutritive derangement (anæmia) from inflammation of the pericardium (pericarditis, etc.); 3, diffuse miliary foci of fat in the sub-endocardial muscular layer of hearts affected with chronic excessive dilatation of the ventricles. In such cases the degenerated muscular tissue will become perceptible through the attenuated endocardium, especially at the trabeculæ and papillary muscles near the apex of the heart. It will appear whitish, yellowish, speckled or striped; 4, larger centra of lipomatosis of an inch or more in diameter, most frequently found in the apex of the left ventricle, or in the midst of the muscular tissue near it. The great friability of the tissue leads very often to rupture of the wall, but differs from ruptures occurring in degenerated myocardia, in this, that here the muscular tissue is separated in layers and the blood infiltrated between them. (*vide* Sect. II, Tab. V, Fig. 3.) The most frequent cause of such lesions is found to be an atheromatous state or thrombotic occlusion of the larger branches of the coronary arteries.

Fatty degeneration is chronic in its nature. Its most obvious causes appear to be valvular diseases of the heart; pulmonic emphysema, general systemic anæmia, or only cardiac anæmia. A destruction or transsection of the cardiac branches of the pneumogastric may also, according to Eichhorst, produce fatty changes of the heart. Infectious and toxicæmic diseases may also superinduce fatty changes, and be followed by albuminous turbidity, whereby the muscular tissue will assume a peculiar grayish discoloration. The cells will appear as if covered with dust, and the albuminous granules will disappear on addition of some acetic acid. Heart rupture frequently forms the termination of such a lesion.

Fatty degeneration of the endocardium must be distinguished from the above disease of the muscles. The endocardiac lesions are generally found as minute, circumscribed foci, forming opaque white spots, and consist in a fatty change of some of the endocardiac connective tissue. Fatty spots are very often formed on the auriculo-ventricular valves (especially the mitral). They are sometimes minimal in size, sometimes occupying the larger portion of a valve. It is essentially a senile derangement, but does not spare younger, but weaker persons.

Amyloid degeneration may affect the connective tissue of the organ. Miliary spots, recognizable only by the microscope and reagents, are most frequently met with. Larger spots are very rare.

It often happens that the connective tissue of the endocardium, myocardium or pericardium becomes sclerotic. This alteration may be found in the shape of flat spots or in rugous elevations, giving the valves a granular appearance. Wherever this process extends the muscular tissue becomes atrophied. It differs from ordinary connective tissue by being more transparent and more consistent.

The endocardium often undergoes mucic or hyaline change. In old age this membrane, especially near the valves, becomes sclerotic. This is due to an increase of its connective tissue. The papillary muscles, as well as the valvular flaps, become covered with excrescences and prominences. The nodules of the semi-lunar valves of the aorta may also be thus enlarged and thickened. With the increase of volume there will also take place a change of structure. The fibrinous arrangement will yield to a more homogeneous texture, and may resemble amyloid change, or may really find place by the side of this. These changes may eventually lead to necrobiotic destruction. The mass may become turbid and form a granular conglomerate of structural detritus. Fatty change often precedes the necrobiotic, and there are deposited upon them calcareous crystals. Such a state of the valve has been called athercomatous. The tissues assume an opaque white color, and when there is deposit of lime salts in the tissue it becomes stiff and of a brilliant white color. In extreme old age a peculiar kind of heart softening is met. The tissue becomes transparent and soft, like a jelly. It is mostly located in the mitral and aortic valves (Sect. II, Tab. VIII, Figs. 1, 3, 4 and 5. In mucic change the tissue becomes semi-liquid or sometimes like thin mucus. Associated with these changes inflammatory processes very frequently happen to exist. They lead to infiltration of the altered tissue with small cellular elements. Neoplasmata are also found in it.

Myomalacia is a name given to a condition of softening of the myocardium consequent upon arterial anæmia. Such anæmiæ are the most frequent results of sclerosis and atheroma of the nutrient arteries of the organ. Emboli seldom produce this form of lesion. In the earlier stages of the disease the softened portions are mostly yellowish white and friable. When the softening has so far progressed as to allow extravasation to take place from the capillaries of the obstructed arteries, the infarcts formed in the tissue produce in their surroundings either a dark red or yellowish and brownish spotted appearance, which will gradually turn grayish, grayish-yellow or a rusty color (Sect. II, Tab. VI, Fig. 2), and will become somewhat depressed. The apex of the left ventricle seems to be the most elective locality for this lesion, although found in other portions of the heart. The papillary muscles may also become the seat of such softening and assume a grayish yellow or hæmorrhagic appearance. When the intima of the heart becomes affected with such a lesion, the thrombi will form polypi upon its surface. When the whole thickness of the myocardium becomes softened, ruptures of the tissue with more or less severe hæmorrhages into the pericardial spaces will take place. The rent thus formed is generally of an irregular, zig-zag shape. (Sect. II, Tab. V, Fig. 5.) The histological changes taking place in this lesion are partly of a progressive and partly retrogressive nature. Ischæmia is the first cause of the muscular atrophy and decay. The muscular fibres undergo partial destruction and form a granular detritus. The connective tissue of the bundles undergoes the same granular changes.

When hæmorrhages are connected with the decay of the tissue, blood corpuscles may be found in the decayed structures, which will leave, after their partial absorption, pigment granules. When the decay reaches this grade of disintegration of the cardiac tissues, death more or less sudden takes place. Should it not end fatally, the process of repair begins by absorption of the detritus, and cicatrization ensues. A reactive inflammation sets in, the blood vessels adjacent to the wound dilate, and emigration of corpuscles, which use up the remnants of living substance still left in the injured tissue, will take place, and cicatricial connective tissue will become organized. The muscular tissue is forever lost, and the replacing structure contains but few cellular elements. What little muscular substance is left alive remains imbedded in the cicatrix. This also incloses the hæmorrhagic detritus, such as pigmentary material, blood crystals, etc. The appearance of such tissue is at first of a transparent gray and afterwards a glittering white (Sect. II, Tab. VI, Fig. 2. Sect. II, Tab. IV, Fig. 2). "The reconstructed portion of the heart never reaches the volume of the former normal tissue but remains as a thin but very unelastic and firm portion of the wall." (*E. Ziegler Pathol. Anatom., 1883.*)

"Myomalacia has hitherto been but little studied, for a number of its phenomena being common to many other diseases of the heart, it was confounded with those. Yet it is essentially a lesion of a special kind, constituting a peculiar form of anæmic necrobiosis. Fatty degeneration and inflammatory phenomena, though occurring in this disease, are not necessarily manifestations of its process, but are only secondary in the chain of phases of its development. It resembles in its mode of proceedure that of softening of the brain, and is not at all a rare disease. Recently Tautain, in his *These de Paris 1873* and *Laveran Union Medical 1878, No. 27*, have clearly described it."—(*Ziegler Patholog. Anat., Vol. II*, p. *50.*)

Samuelson, Conheim and Schulthess have in their experiments proven that stoppage of circulation of the coronary arteries produces, shortly afterward, a fall of aortic pressure and cessation of the heart's action in a state of diastole. But as the anæmia found in diseases of the heart is slowly and gradually produced, and likely exists only in circumscribed portions of the organ, the clinical phenomena of the affection will only be: temporary incompetence of the heart's action, stenocardia and blood-stasis in the lungs.

ENDOCARDITIS.

Acute endocarditis is an inflammatory process caused by a certain phlogogenic substance or substances circulating in the blood. The valves are oftenest affected, although any other portion of the inner surface of the heart may become involved. The most frequent form of this inflammation is the one called *verrucous*, and is manifested by formation of miliary or even larger nodules projecting above the endocardial surface. These may have a transparent or opaque grayish white or yellowish white appearance. They are generally covered with white or grayish red thrombi, and only become visible after removal of the thrombi. When situated on the valves, these excrescences often form rows corresponding to the margin of the valvular flaps. At times they are disseminated over the largest portion of the valve and often form quite extensive groups of wart-like prominences. (Sect. II, Tab. VIII, Figs. 1, 2, 3.)

Situated elsewhere, these arrangements are not quite so regular. When the prominences are very small but very numerous, and close to one another, they give the surface a velvety appearance. They

TABLE VI.

PRIMARY STAGES OF MYOMALACIA.

FIG. 1.–*Hypertrophic papillary muscles in the left ventricle.*

A longitudinal section of the ventricle shows its walls to contain numerous solid infarcts. Towards the apex the muscular tissue had undergone fatty and atheromatous change; a portion of the myocardium looks spotted grayish yellow (*P. R. I.*), some white fibers binding the edges of the altered tissue.

Outside, the heart has the appearance of a fatty heart. Whilst the hæmorrhagic portions look suffused with blood, the balance of the heart's tissue looks paler than usual. In this stage of the lesion the heart is still capable of contraction. The embolic condition of the coronary artery branches have given rise, in this case, to stagnation of the circulation in the muscular tissue, rupture of the small vessels and extravasation into the wall of the ventricle. The apoplectic foci are adjacent to and within the limits of the degenerated myocardiac structure. (*F. A.*, apoplectic focus; *V. D.*, right ventricle; *V. S.*, left ventricle; *A. A.*, arch of the aorta; *V. P.*, pulmonary artery.

FIG. 5. *Later stage of Myomalacia.*

The left ventricle is considerably dilated in a longitudinal direction. Portions of the wall and the apex have undergone connective tissue degeneration in the process of cicatrization; that portion of the endocardium is of a grayish white color, perfectly anæmic and much tougher than usual. A large clot is lodged in the apex and is surrounded by the degenerated tissue. It constitutes what is ordinarily called a partial aneurism. The connective tissue between the muscular layers of the apex is obliterated, and the layers have lost their cohesion, so that blood readily infiltrated between them and held them separate. In this manter the apex became more dilated and attenuated.

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are sometimes so small as to escape notice, but are readily noticeable by the microscope. The excrescences are formed by subendothelial exudation. They may have a granular or fibro-granular texture. In the granular masses many pale lumps are to be seen; they seem to be endocardial cells undergoing gangrenous coagulation. The inflammation takes place in the tissue, and the exudates seem to enter it and then become coagulated. The surrounding tissues of the coagula become compressed and mortified, but those simply infiltrated with small spherical cells retain their vitality. "This form of inflammation belongs to the group of diphtheritic lesions, and resembles mostly, in its histological character, superficial diphtheria of mucous membranes and the second stages of variolous development."—(*Ziegler Patholog. Anatom.*)

More mitigated varieties of endocarditis exist, in which there is no formation of vegetations, and only consist of cellular infiltration into the connective tissue of the endocardium.

The cause of the inflammatory exudation is, according to Klebs and Eberth (*Archiv. for experim. pathol. and Virchow. Archiv.*), an invasion of the endocardium by microorganisms, at least in a great number of cases. Ziegler found in a case of highly acute verrucous endocarditis, which led to metastatic inflammation in the muscular tissue of the heart, and in the parenchyma of the kidneys, a great number of micrococci in the blood vessels of those organs. Nauwerk, in Ziegler's pathological laboratory, similarly found micrococci in the inflamed endocardium; they existed not only in the hypertrophied valves, but also in the unaltered portions of the endocardium and myocardium. In rheumatic polyarthritis it forms the most frequent complications. In typhus and typhoid fevers of the graver sorts, in general suppurative ulcerations of the tissues, in carcinomatous and kindred diseases, it exists as a part affection. Leyden found it in chronic suppurative gleet. Taken altogether, its causes are many, but they seem to be more connected with microparasites than with anything else. Rosenbach (*in Arch. fur experim. pathol.*) in his article on valvular lesions artificially produced, correctly observes: "How uncomplicated endocarditis in man is produced I do not venture to say; anyway it must be clearly distinguished from *bacteric endocarditis*. There is indeed no more reason for the non-existence of such uncomplicated lesion than for non-existence of simple pleuritis, which nobody would ever think of attributing to infection. On an inflamed valve, vegetations form very readily."

The subsequent results of endocarditis vary according to the nature of the individual cases. The upper layers of the vegetations are never organizable. Small portions may become absorbed; now and then they undergo a calcareous change. Most frequently they decay and form detritus, parts of which are carried away by the blood. Calcified masses may also pass into the blood current. But both lead to formation of emboli.

Loss of tissue is produced by removal of dead substance resulting from ulceration, which may be slight or very extensive. In the so-called ulcerative endocarditis the destruction penetrates deep into the cardiac substance, and an extensive portion of the valve will be deeply ulcerated. The enfeebled and inflamed structure of the valve will be dilated and acute valvular aneurism will result. Should the destructive process continue still farther, perforation of the valve with loss of considerable portions of its tissue will be the result. The same ulcerative destruction may happen in the chordæ tendineæ, and they may be torn from the flaps. The ulcerative form of endocarditis is especially found in inflammation, having its origin in pyæmia or septic infection. Verrucous and ulcerative endocarditis differ only in the causes of their production, but not anatomically. The pernicious or ulcerative form seems due to pyæmia, whilst the verrucous is due to rheumatic arthritis.

When the acute stage of inflammation does not lead to death, then the productive inflammatory process, which leads to formation of new connective tissue and cicatrization, follows the exudative and gangrenous disintegration. The tissue which has not been gravely involved is the one from which the reparative work proceeds. For weeks and months the hyperplastic tissue may be found in a state of quasi granulation. Migratory leukocytes, from the vessels, and cells produced by proliferation of the fixed tissue, very likely constitute the elements of newly-formed structures. Large numbers of new vessels supply it with nutrition. By this plastic process the endocardium becomes considerably hypertrophic. The neoplasmata appear on the walls of the auricles and ventricles as fibrous, white, transparent spots, sometimes disseminated and sometimes confined to certain localities. The valve flaps are thickened, sclerotic, retracted and grown together, and deformed in various ways. The chordæ tendineæ are likewise shortened, thickened and also grown together. Within the hyperplastic structures all sorts of degenerative processes are carried on. Atheromatous and lipomatous degenerations are the most frequent. All these changes render the valves more or less incompetent to perform their work of closure of the ventricles during systole. The sclerotic and hyperplastic processes lead to constriction or stenosis of the opening, whilst the retraction and deformity of the flaps and the tendinous cords produce incompetency (insufficiency) and inability of complete closure. Stenosis and incompetency are very often associated. The number of grades, possible to exist in this class of deformities, is too great to be described in detail. The consequences of such valvular diseases have been described above. Of course when the blood cannot readily pass the orifices and the ventricles emptied, it will regurgitate and be dammed up in the portions of the circulation back of the valve, and dilatation of these parts will ensue. For the purpose of compensating for this disturbance, the portion of the muscular tissue of the heart which has to propel the stagnant blood will become hypertrophic. Frequently the inflammation will extend from the endocardium to the tissues situated beneath, or it will begin beneath and extend to the endocardium; either way there will be complications. The histological character of this form of inflammation is distinguished by infiltration of small cells into the tissues.

The anatomical alterations taking place in this form of myocarditis are either induration or formation of abscesses. In the first case hyperplastic connective tissue will form what is called a cardiac callosity. It consists of white, glistening fibrous strings or cords. The endocardium will contain spots of similar appearance and consistence. The muscular bundles are frequently thus altered. Such small callus will never show any derangement of function, provided there are not too many. Larger ones will prevent the complete contraction of the heart. Should they be large and yielding, they will gradually form a diverticulum in the cavity, and a so-called aneurism of the heart will be produced.

Purulent myocarditis results from pyæmic infection, and is brought about by bacteria carried by the coronary arteries into the myocardium. There are formed yellowish white or grayish abscesses. When these break open, ulceration of the heart will be the result; should they break through externally, purulent pericarditis will follow. Extensive myocarditis often causes rupture of the heart. (*Vide* Sect. II. Tabs. 4, 5.)

The causes of myocarditis are generally the same as those of endocarditis. Leyden found myocarditis frequently to follow diphtheritis. Rosenbach found in cases of diphtheria granular concrements of the heart in a state of waxy degeneration. Small pox, epidemic meningitis and recurrent typhus are, according to Leyden, often complicated with myocarditis.

The most frequent cardiac infectious granulomata are those of a tuberculous, seldom of a syphilitic nature. The first are generally complications of general tuberculosis, and are mostly found in the endocardium of the right ventricle. Large caseous masses are more seldom than miliary tubercles. They are generally sequellæ of pericardial *caseation*. Rare gummatous nodes are found imbedded in hyperplastic connective tissue in the cardiac walls, in the shape of gray or grayish red foci, either as soft, or dry and friable masses. More frequently than these, sclerotic inflammations of the myocardium are to be found in hereditary syphilis. Primary fibrous, lipomatous, myxomatous or sarcomatous tumors proper are of very rare occurence in the heart. The most frequent are the carcinomatous secondary deposits, produced by infections from other parts of the body. They may occupy any place in the heart.

The results of tumors (outside of the malignity of their nature) always vary according to their size and the position they occupy in the heart, and may range from the very mildest to the most pernicious grades of injury to the organ and to the body.

GENERAL PATHOLOGY OF THE PERICARDIUM.

Inflammation of the pericardium, the most important of its diseases, is connected with exudation, which like those of any other serous membrane, may assume a great variety of forms; and these may undergo manifold changes, some of which may lead to the destruction of the person affected, or may not in the least endanger the life or even the health of the person.

Conheim in his classic researches into the process of inflammation has graphically set forth the successive phases of inflammation of this serous membrane, which are, shortly, as follows: The first stage consists in the formation of an exudate of serum, containing cells, which collect within the tissue itself and on its surfaces, and is followed by partial disquamation and destruction of the epithe-

EARLIER AND LATER STAGES OF MYOMALACIA.

Fig. 1.

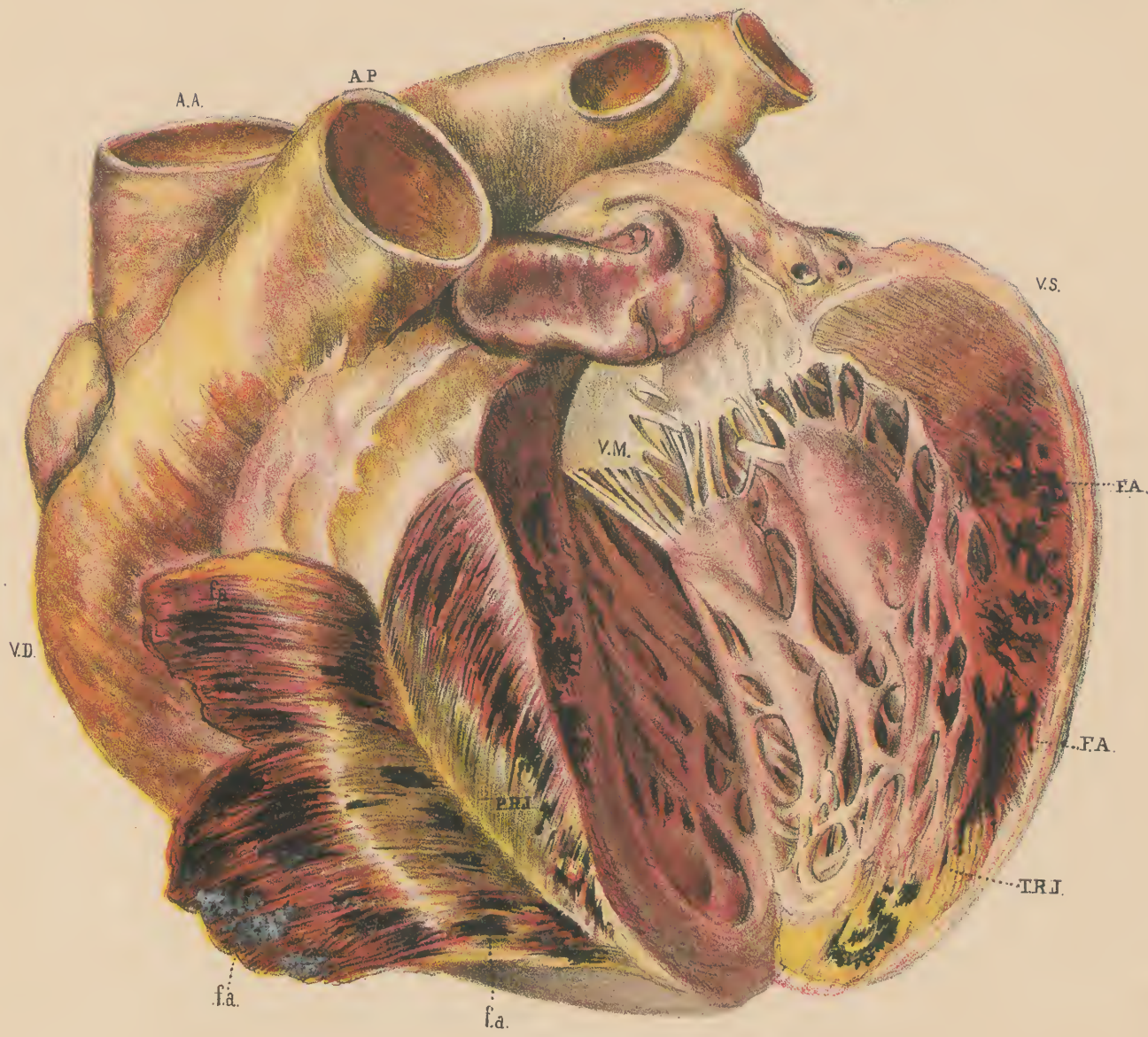
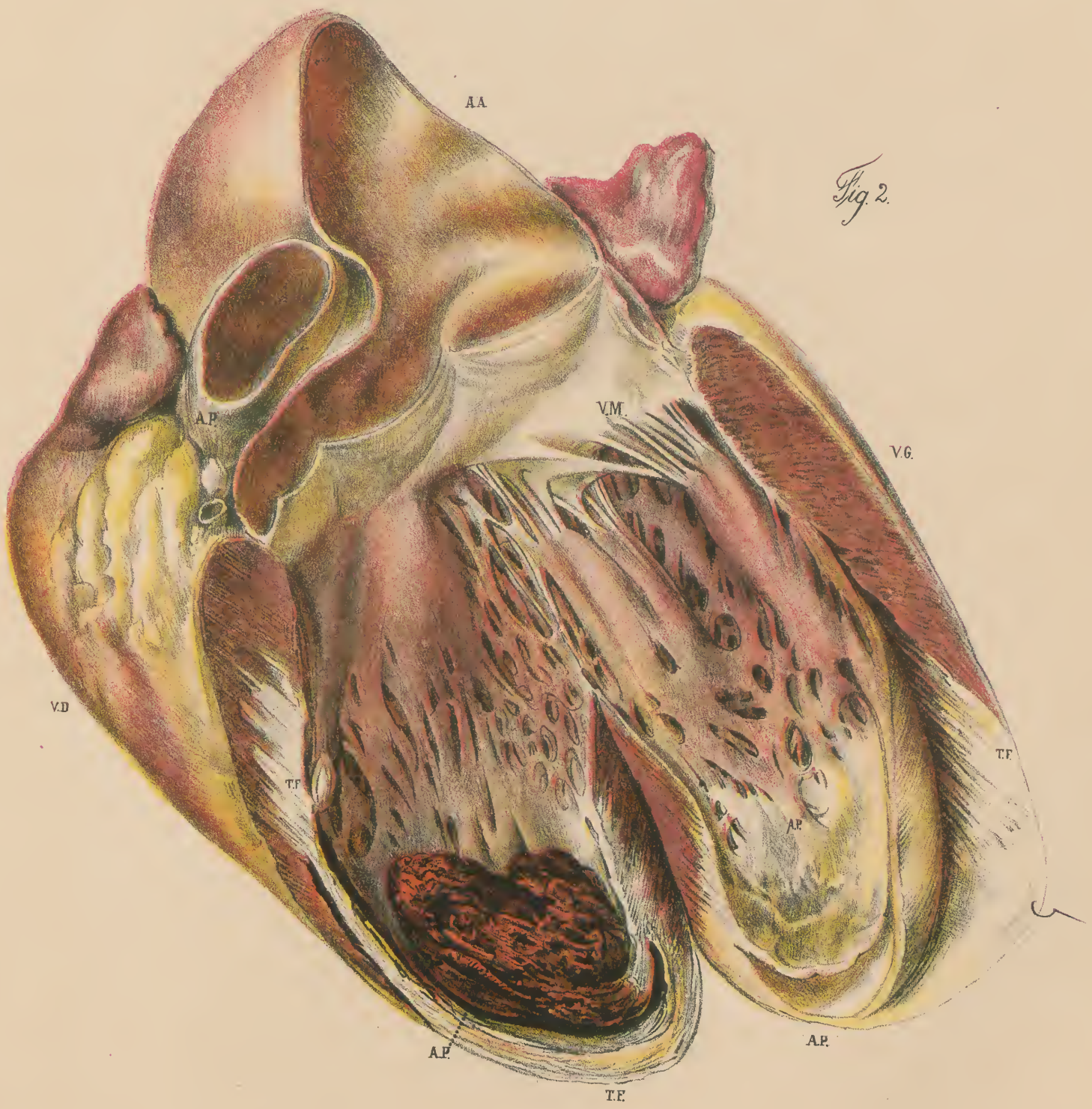


Fig. 2.



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Sec. II. *Tab. VII.*

ULCERATION OF ENDOCARDIUM.

PARTIAL ANEURISM OF LEFT VENTRICLE.

Fig. 1.

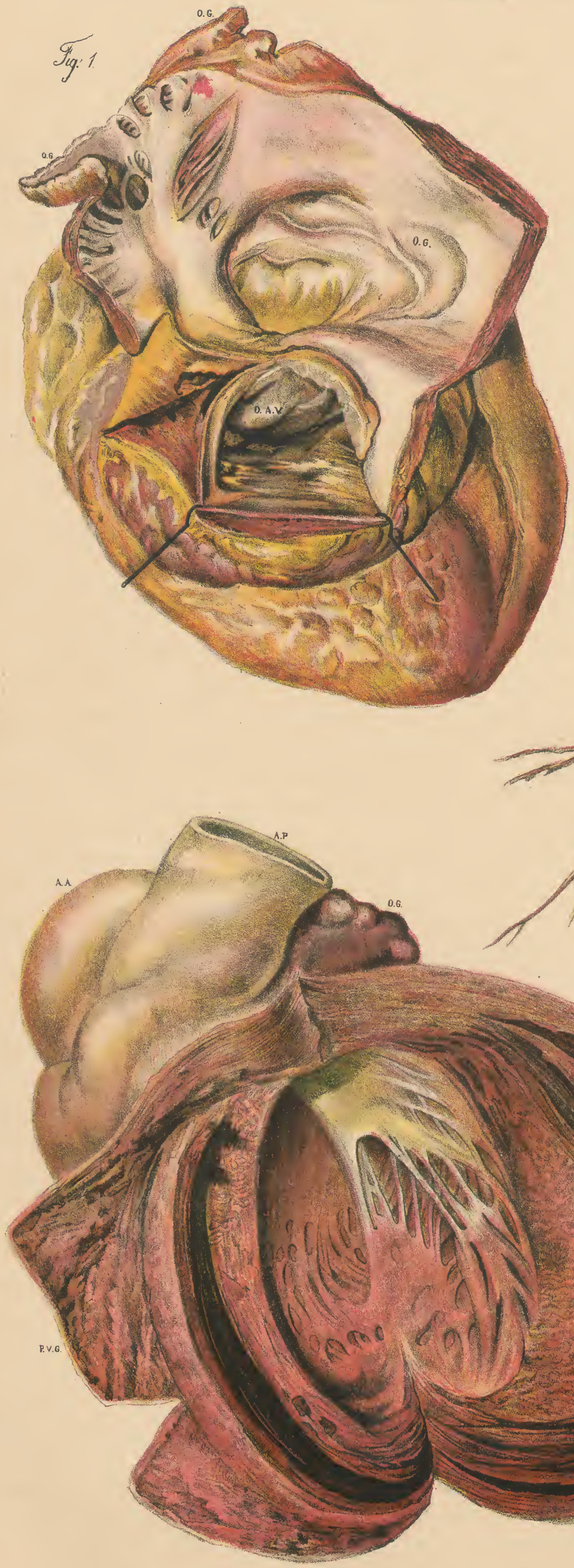

Fig. 2.

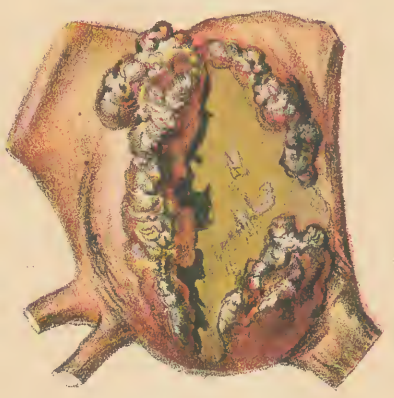

Fig. 3.

Fig. 5.

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TABLE VII.

FIGS. 1, 2, 3.—*Ulceration of the Endocardium.*

CASE.—A girl of sixteen years. Was primarily affected with furunculosis. She was suddenly attacked by a violent fever, with temperature of body 106 deg. Fahr., and upward; coma and death soon followed. During life there was nothing apparently abnormal in the heart.

Post-mortem—showed a great quantity of postules scattered over the body; besides quite a number of hæmorrhagic spots in different parts of the bodily tissues. Miliary hæmorrhagic foci in the brain, spleen, liver and intestines; infarcts in the lungs; in the muscular tissue of the heart, a number of soft, yellowish-gray incrustations and irregularly-formed yellow spots; upon the mitral valve a ring of ulcers (Fig. 2) situated on the basal portion of the flap. The right segment nearly destroyed by confluent ulcers (Fig. 3); the tissue of the flap infiltrated with blood; at the bottom of the ulcers, yellowish-gray vegetations; on the endocardium of the left auricle, yellowish ulcerative masses (Fig. 1, *O. S.*); the yellow efflorescences on the mitral valves consisted chiefly of masses of zooglœa of spherical bacteria, which were readily made visible by dissolving the commingled fibrin with glacial acetic acid; in the kidneys, especially on the pelvic surface, a great many yellow and gray nodules and hæmorrhagic spots; in all metastatic deposits great quantities of bacteria. (*O. S.* left auricle; *O. A. V.*, auriculo-ventricular opening.)

FIGS. 4, 5.—*Partial aneurism of the left Ventricle.*

The ventricular cavity laid open, shows the endocardium to have assumed a striped and superficially wavy appearance. Portions of the ventricular wall are infiltrated with blood, the muscular layers separated, and the intermuscular spaces filled with clots; smaller infarcts visible here and there. Fig. 5 shows the brownish color of the muscular tissue and furrows made by abscesses and corrosive pus, in which the blood ultimately entered, and caused dilatations; the endocardium as well as the pericardium have both been involved in the morbid processes; the pericardium attached to the heart, and the endocardium covered with many ridges of newly-formed connective tissue.

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lium. Only in the mildest form is the epithelium preserved intact or only proliferates.

There are three principal forms of inflammation: fibrinous, sero-fibrinous and purulent. The fibrinous form is also called adhesive.

FIBRINOUS FORM.

In recent fibrinous and sero-fibrinous inflammation the membrane is more than usually injected, its surface turbid and lackluster, which is due to a deposit of coagula. The first fibrinous deposits are generally in the shape of little lumps, composed of strings and conglomerate. The epithelium, in milder forms, remains uninjured between the fibrinous coagula; in more intense forms it is transformed into a homogeneous or granular, loosely adherent membrane. The deposits form without any considerable quantity of serum in its cavities. It is then called *pericarditis sicca* (dry pericarditis), and often leads to synechia of the serous plates. When the exudation is more abundant, there is more infiltrate in the serous cavities. Under such circumstances the pericardium will be distended at the expense of the lung, which will become compressed, if the exudation extends to the pleura. The exudate contains variable quantities of cells and fibrin; colorless and colored blood corpuscles are also contained in it in variable quantities. The fibrin is precipitated upon the membrane and forms layers of different thicknesses. Upon the surface of the heart the deposit forms prominences of many forms (cor. villosum), as described in Sect. II, Tabs. 1, 2. Such villosities always lead to attachment of the plates. The extent of the inflammation varies greatly; attacking sometimes a very small portion of the membrane and sometimes its whole surface. After the exudate has reached its maximum, it stops, and commences to undergo reparative processes, which consist at first in absorption of the exudate and development of new connective tissue. Of course, the process of absorption will vary greatly in time and extent. The fibrinous deposits, though very slow to disappear, will often be dissolved and carried off. The residual portions often become calcareous. The presence of fibrin, which may be considered as a foreign body within the living organism, will produce inflammatory neoplasmata. A few days after the beginning of the pericarditis, a number of small vessels will form upon the surface below the fibrinous deposit, and enter into the pseudo-membrane, which will indicate a beginning of organization of tissue. In its deeper layers a great number of migratory cells will be found, and will constitute the basis of the new connective tissue.

The effect of the inflammatory tissue-formation is variable; when of slight extent, it forms a slight induration of tissue in the shape of white, glistening spots. When very extensive, thick fibrinous, or even cartilagenous plates or extensive callosities will result. This form of pericarditis may take place in the membrane alone or in conjunction with endocarditis or myocarditis, or as a secondary process of pleuritis or mediastinitis. The whole membrane, or only a portion, may undergo this inflammation. The heart may be free or attached. This attachment may be by means of a few strings or fibers, or the whole organ may be held by very many such strings or cords. The consistence and solidity of these attachments do also greatly vary. They sometimes remain soft and pliable, or they may be calcarized, and the whole may be enveloped by many plates of salts of lime.

Purulent pericarditis may be a primary process or a secondary development of a sero-fibrinous inflammation and an enormous emigration of colorless corpuscles.

Purulent inflammation often exists simultaneously with fibrinous, and the pus corpuscles may be surrounded by fibrinous coagulæ, and form pseudo-membranes, containing large quantities of pus. Such an exudation may assume a putrid or septic character, and contain enormous numbers of microparasites. The pericardium, in any of these kinds of inflammation, is highly infiltrated with small cells, highly injected and denuded of its epithelium. In uncomplicated purulent exudates, the pus corpuscles undergo early, fatty change and disintegration. However, purulent exudate is far slower absorbed than serous. Generally, death takes place at the height of this form of inflammation. Frequently only the liquid portion of it is absorbed, whilst the fatty degenerated corpuscles become inspissated into a caseous mass, which either remains permanent, or is very slowly dissolved and re-absorbed. Very often extensive quantities of pus on the pericardial surfaces lead to formation of granulations and connective tissue, which may become, under circumstances, exceedingly extensive.

Tuberculosis of the pericardium may be of three different origins:

1. Part affection of general systemic miliary tuberculosis. 2. Infection from an adjacent structure or physiologically-affiliated tuberculous tissue. 3. A tuberculous focus in the pericardium itself.

Tubercles may exist in the membrane without any concomitant inflammation, and characterize the miliary form; or there may be extensive inflammatory changes co-existent, which will lead to formation of liquid exudates. Then, also, the diffusely-disseminated changes produced by the inflammation may form the substratum to the tuberculous formations, and the tubercles will be imbedded in such neoplasmata. There are no defined limits to either of the anatomical modifications. They may all exist by the side of one another. Gray colored miliary eruptions, without local inflammation, very rarely take place in the pericardium. With moderate inflammation they form a derivative of infections in the shape of congeries of small cells surrounding the vessels. They vary in extent to a high degree.

The most frequent form of tubercular pericarditis is manifested as a total adhesion of both plates; sometimes enclosing in the uniting mass quantities of liquid, cellular or fibrinous exudative substances.

Tumors or cysts of any kind may be said never to develop primarily in the pericardium; secondary heteroplasia occur now and then by metastatic processes.

The clinical character of pericarditis is of a far less satisfactory nature to the physician than its pathology, for as a part affection of very many inflammatory and infectious diseases, it is so often covered by morbid phenomena of those to which it is only secondary, or with which it is pathologically connected, that it requires most favorable circumstances to manifest pericardiac symptoms in their purity; for idiopathic pericarditis is one of the rarest diseases of this serous sac. It is not any less important, for that matter, for many of its lesions are of such a gravity, and their results of such consequence, that, like the diseases of the heart proper, with which they are usually associated, they have to be thoroughly understood, and often directly treated. As stated above, pure and simple pericarditis very often passes unperceived by the patient and his medical attendant. Only the graver kinds, complicated with massive exudations, or when associated with myocarditis, endocarditis, diffuse pleuritis, pneumonia, pleura-pneumonia, and the many forms of infectious and inflammatory polyarthritis, come in for a share of the physician's attention. Dyspnœa and pain are two of the chief symptoms in the early stages of pericarditis, before exudation has so accumulated as to show clear physical signs by inspection, auscultation and percussion. Wertheimer in (*These de Paris*) has qualified the pain as either central or peripheral. The peripheral, due to the irritation caused by the inflammation of the membrane upon the terminal filaments of the phrenic and intercostal nerves. The pain is found below the nipple, in the pericardiac region, in the epigastrium, sometimes situated on the left side of the xiphoid cartilage, or extending to its neighborhood, to the base of this cartilage, to the lower portion of the neck, at the origin of the sternomastoid muscles. It may be produced by pressing upon the parts, or may exist without pressure. The central pain is due to irritation upon the pneumogastric. It is very intense, and will give rise to dyspnœa and angina pectoris. Central pain is indicative of a dangerous condition; peripheral, generally, forbodes no great danger. Painful pericarditis is either of a traumatic origin, or idiopathic; secondary pericardial inflammation is developed without causing pain.

Next to the exudative pericarditis, the symptoms of which are nearly all of a physical nature, and have been described, in their main features, in the first part of this section, the adhesive form is one of the lesions of this membrane, which mostly requires the physician's attention; especially when the symphytic process is very extensive, and reaching far and deep into the adjacent vital organs. But its diagnosis forms a weak feature in its clinical character. In but few cases can it be recognized with certainty. The reliable symptoms are: distinct depression or retraction of the sternum and a large portion of the lower part of the thorax, diastolic collapse of the veins of the neck, systolic depression of the intercostal space, corresponding to the apex-beat; so-called pulsus paradoxus, etc. Ries (*in Berlin Klinische Wochenschrift*) calls attention to a symptom which, although of rare occurrence, is yet very important, that is, metallic sound of the heart region, produced

TABLE VIII.

FIGS. 1, 2, 3, 4, 5, 6.—*Stenosis of the left auriculo-ventricular orifice, hypertrophic sclerosis of the mitral valve.*

A number of pockets opening into the ventricular cavity. FIG. 1. Left auriculo-ventricular opening as seen from the auricle (*A. O. V.*) FIG. 2. The same as seen from the ventricle. FIG. 3. The mitral valve spread out by a vertical section upon the ventricle, the auricle and valve flaps. FIGS. 1, 2, 3. Show the cavities or pockets formed by ulceration and neoplastic formations upon the valvular surfaces. 1, 2. 3, 4, *K.* Show the pockets or cavities opening into the ventricle. 1 *K.*, FIG. 2. is so large as to intercept a portion of the blood passing through the orifice. The others offer more or less resistance to the passage of the blood from the auricle to the ventricle. FIG. 4. Shows ordinary stenosis of the mitral valve. The two flaps thickened and contracted, leave but a narrow opening (*O. A. V. G.*) for the passage of the blood. A number of chordæ tendineæ, also shortened and thickened, are fixed here, and to them are attached the columnæ carneæ of the left ventricle. FIGS. 5, 6. Show a rather unusual specimen of stricture of the orifice. Seen from the auricle, FIG. 5, the auriculo-ventricular opening looks as if lined with a number of tubercular prominences, partly free, partly covered by the endocardium. The orifice is so narrow as not to allow the small finger to pass through it. FIG. 6. Shows the ventricular surface of the orifice (*O. A. V. G.*) The mitral valve itself is less altered than its adherent border. Many verrucous prominences exist upon the valvular surface.

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by gastric consonance. His explanation of this metallic sound phenomenon is, the very close approach of the heart and diaphragm to the gastric wall. *Ebstein* and *Leichtenstern* have also described these metallic sounds of the heart in adhesion. *Kussmaul* found in certain cases of mediastino-pericarditis the pulsus paradoxus and inspiratory enlargement of the veins of the neck. This pulsus parodoxus is characterized by lessening the caliber, or completely obliterating the arterial pulse during inspiration. He ascribes it to attachment of the pericardium to the sternum. The extent of dullness on percussion in adhesion is very variable, sometimes very extensive, and sometimes but slightly perceptible. This variation is mainly due to the amount of displacement of the heart.

The older writers held that long-continued extensive pericardial adhesion led to hypertrophy of the heart, but later writers have nearly all rejected that opinion, and claim that when the heart is surrounded by very strong and unelastic large bands, the heart's tissues gradually degenerate and become atrophic, but not hypertrophied. Exocardial or pericardial murmurs are always friction sounds; they are due to roughness or dryness of the surfaces of the serous plates. Very rarely are they produced by excessive enlargement of the heart. By far the greatest number of those sounds are in consequence of pericardiac inflammation. The variety of these murmurs is great, sometimes very harsh, and sometimes very soft, so as to imitate endocardial murmurs. The differential murmurs (pericardial and endocardial) are discernible by their relation to the rhythm of the heart. The endocardial are either diastolic, presystolic or systolic. The pericardial follow no rhythm. Slight pressure upon the chest with the stethoscope often increases the intensity of the pericardial murmur. Endocardial murmurs are not influenced by moderate pressure, whilst strong pressure upon the thorax causes them to disappear altogether. Inspiration, as a rule, increases the intensity of pericardial and endocardial murmurs. There are some exceptions to this.

Endocardial murmurs extend far beyond the cardiac region of dullness; pericardial are strictly confined to that region. Changes of posture modify pericardial murmurs far more than endocardial. Especially the upright position with the body bent a little forward. Pericardial murmurs appear as coming from the surface; endocardial as coming from a certain depth. Pericardial murmurs are very changeable; endocardial preserve their character unchanged. The form of the dullness of the heart-sound determines a pericardial murmur. Pleuro-pericardial murmur is usually heard along the left anterior border of the lung, closely proximate to the heart. It differs from pure pericardial murmur by being influenced, and completely disappearing when breathing is suddenly checked. *Rosenbach* found it to extend sometimes quite a distance from the heart.

A pericardio-diaphragmatic friction sound has been described by *Emminghouse;* it was produced by movement of the heart upon the tendinous portion of the diaphragm, by displacement. The friction sound in exudative pericarditis can, of course, only be heard when there is no great quantity of liquid present between the plates. It is generally audible near the basis of the ventricles and along the left sternal border. Its duration is very variable, sometimes lasting for quite awhile and sometimes quickly disappearing, to reappear again. The intensity of the sound is hardly ever proportional to the amount of inflammation of the membrane. The cause of the friction sound has not been definitely established as yet.

PHYSICAL DIAGNOSIS OF DISEASES OF THE HEART.

Dilatation and increase of volume of the heart are the most important subjects for physical diagnostication of diseases of the organ. Generally both conditions co-exist; for even in simple hypertrophy, the volume of the organ is augmented; the very nature of that process indicates increase of quantity of the heart substance, in whatever portion of the organ it may exist. According as either one or both ventricles are dilated and hypertrophied, the morbid phenomena will differ.

A. Hypertrophy of the left ventricle can be diagnosed by the following symptoms: 1. The apex beat will show great increase of force, whilst its locality and extent will be unchanged; 2. Dull sound on percussion will increase in depth, but neither in extent nor locality; 3. Diastolic sound of the aorta will be stronger, and sometimes will have a peculiar ring; 4. The radial pulse will have a great tension, and will not readily be compressed by the finger. Sclerosis of the arteries and renal atrophy, stenosis of the aortic orifice, compression, constriction or congenital narrowness of the aorta, are mainly the lesions when hypertrophy takes place; for in all of them there are obstacles formed, which only an increased quantity of muscular tissue in the left ventricle can overcome.

B. Dilatation of the left ventricle furnishes the following physical signs: 1. The apex beat passes externally in extent beyond the mammilary line, and is located lower than usual; 2. The area of dull sound of the cardiac region is increased above, below and to the left, and is of oval form; 3. In the cardiac region systolic, diffuse vibrations become perceptible. Uncomplicated dilatation is hardly ever perceptible. Connected with hypertrophy, the vaulting of the thorax is especially noticeable. It is mostly due to incompetency of the aortic valves and aneurism of the origin of the aorta.

C. Dilatation of the right ventricle is manifested by: 1. The cardiac dullness and resistance extending farther to the right than usual; the area of dullness will be of circular form; 2. The apex beat extending to the right. When there are obstacles formed in the pulmonary artery, dilatation of the right ventricle may be expected. The same will generally happen in chronic diseases of the lungs and derangements in the mitral valve and semi-lunar of the pulmonary artery. Dilatation of this ventricle may be formed in chlorosis, long-lasting fevers, and consumptive conditions of the body, when the nutrition of the heart is disturbed.

D. Hypertrophy of the right ventricle can be recognized by the following: 1. Increase of diastolic pulmonary sound; 2. Increase of force of the apex beat, deviating to the right. The causes of this phenomenon are nearly the same as those of the hypertrophy of the left ventricle. When the whole heart is considerably dilated, pericardiac friction sound will appear here and there. Total hypertrophy will very frequently produce the so-called *Cliquetis metallic.* Extensive degeneration and weakness of the heart will yield very feeble, ventricular systolic sounds.

E. Lesions of the endocardium. When there is incompetency of the aortic valves, the following symptoms will naturally appear: 1. Diastolic sound over the aorta. It is mostly audible over the middle of the sternum, and is sometimes continuous over the valves. When some of the flaps are still able to vibrate, there will be, besides the murmur, a diastolic tone. The first sound over the apex is characterized by great weakness, and the first sound over the aorta is absent and replaced by blowing murmur.

2. *Dilatation of the left ventricle* will be brought about by the over quantity of blood entering it, during its diastole (when there is insufficiency of the aortic valves), by regurgitation from the aorta, besides what it receives from the auricle.

3. *Hypertrophy of the left ventricle* must exist when it has to overcome obstacles in the arterial circulation. Such resistances produce increase of compensatory work, and augmentation of muscular tissue in the wall in the same ratio as there exists resistance.

Hypertrophy and dilatation will exist in such a measure as there is insufficiency.

4. *Bounding pulse in the carotids.* Very often there will be felt an arterial fremitus, simultaneous with the cardiac systole; an arterial systolic murmur simultaneous with the cardiac systole, absent during the cardiac diastole. When there is excessive tension in the arterial system, from the great quantity of blood in the aorta, a murmur, simultaneous with the cardiac systole, may be heard in nearly all the middle sized arteries in the head and neck. *Quinke* claims to have noticed, under such circumstances, visible pulsation in the fingertips.

The radial pulse becomes very hard and frequent, and the sphygmograph will show a perfectly vertical rise of the ascension line and formation of a very acute angle in its descent.

F. Stenosis of the aortic opening. This is nearly always associated with incompetency of the aortic valves. In uncombined stricture, the following physical changes must be looked for:

1. *Systolic murmur over the aorta.* It has a peculiar loud scraping character. The diastolic sound, on the contrary, is very feeble; always absent in the carotid and very feeble at the apex.

2. *Hypertrophy of the left ventricle.* Because the narrow aortic orifice offers great resistance to the emptying of the ventricle. In this lesion, the apex beat will be very feeble, notwithstanding the hypertrophy of the walls. *Friedreich* found even systolic depression in such cases. The pulse is found hard, small and slow. The first quality of the pulse is due to the hypertrophic ventricle, the latter to the incomplete filling of the aorta.

G. Insufficiency of the mitral valve will cause the following changes: 1. *Systolic murmur over the apex beat,* extending to the other portions of the heart, and most audible over the opening of the pulmonary artery. Occasionally a systolic sound is associated with it, or may be palpable as a systolic fremitus.

2. *Dilatation of the right ventricle.* The mitral valve, unable to close the ventricle sufficiently, causes regurgitation into the left auricle. This cavity, obtaining more blood than normal, from the veins and ventricle, will become dilated. The left auriculo-ventricular regurgitation will hinder the free forward movement of the blood in the pulmonary artery, and of course dilatation of the right ventricle, as shown above, will follow.

3. *Hypertrophy of the right ventricle* will be produced by the same causes as above, and will be known by an increased force of the second pulmonary sound.

STENOSIS OF LEFT AURICULO-VENTRICULAR ORIFICE.

SCLEROSIS OF MITRAL VALVE.

Sec. II. *Tab. VIII.*

Fig 1.

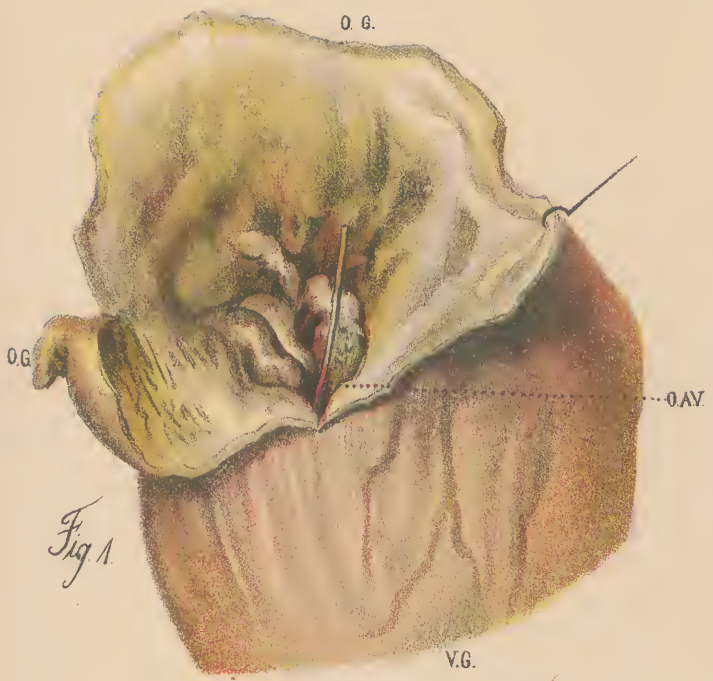
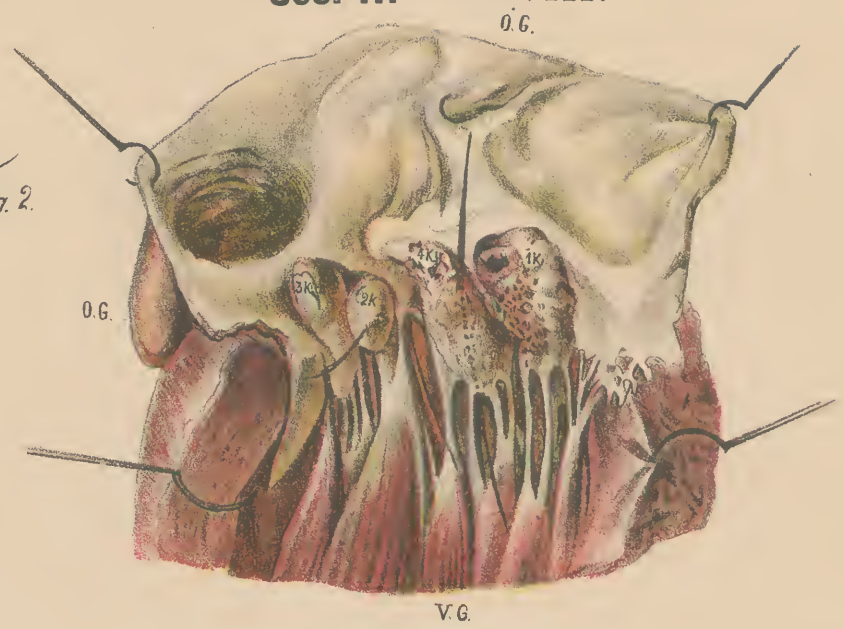


Fig. 2.



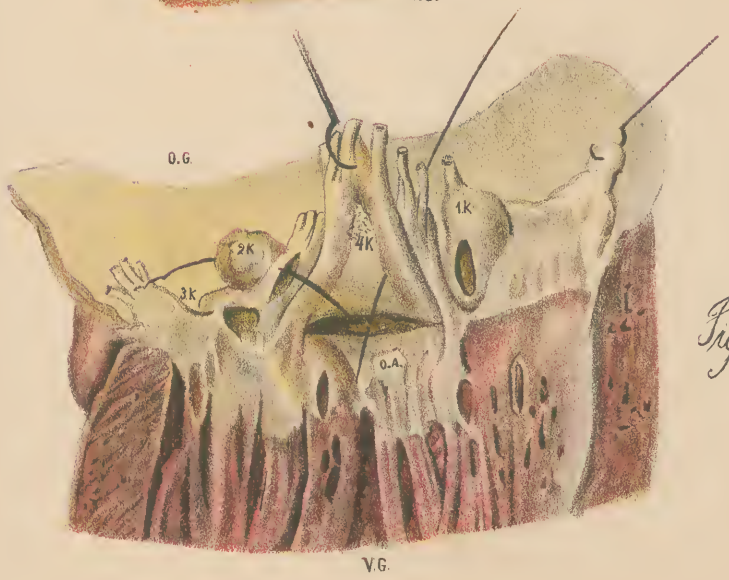


Fig. 3.

Fig. 4

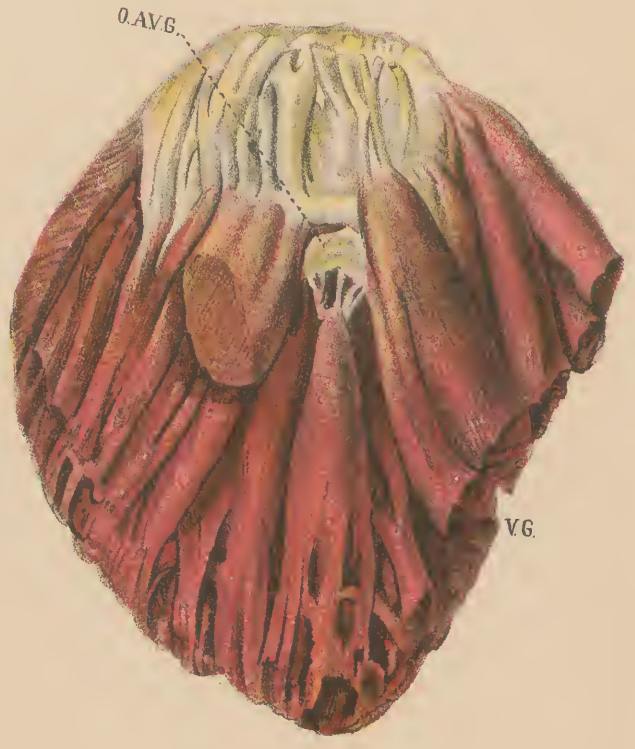


Fig. 5.

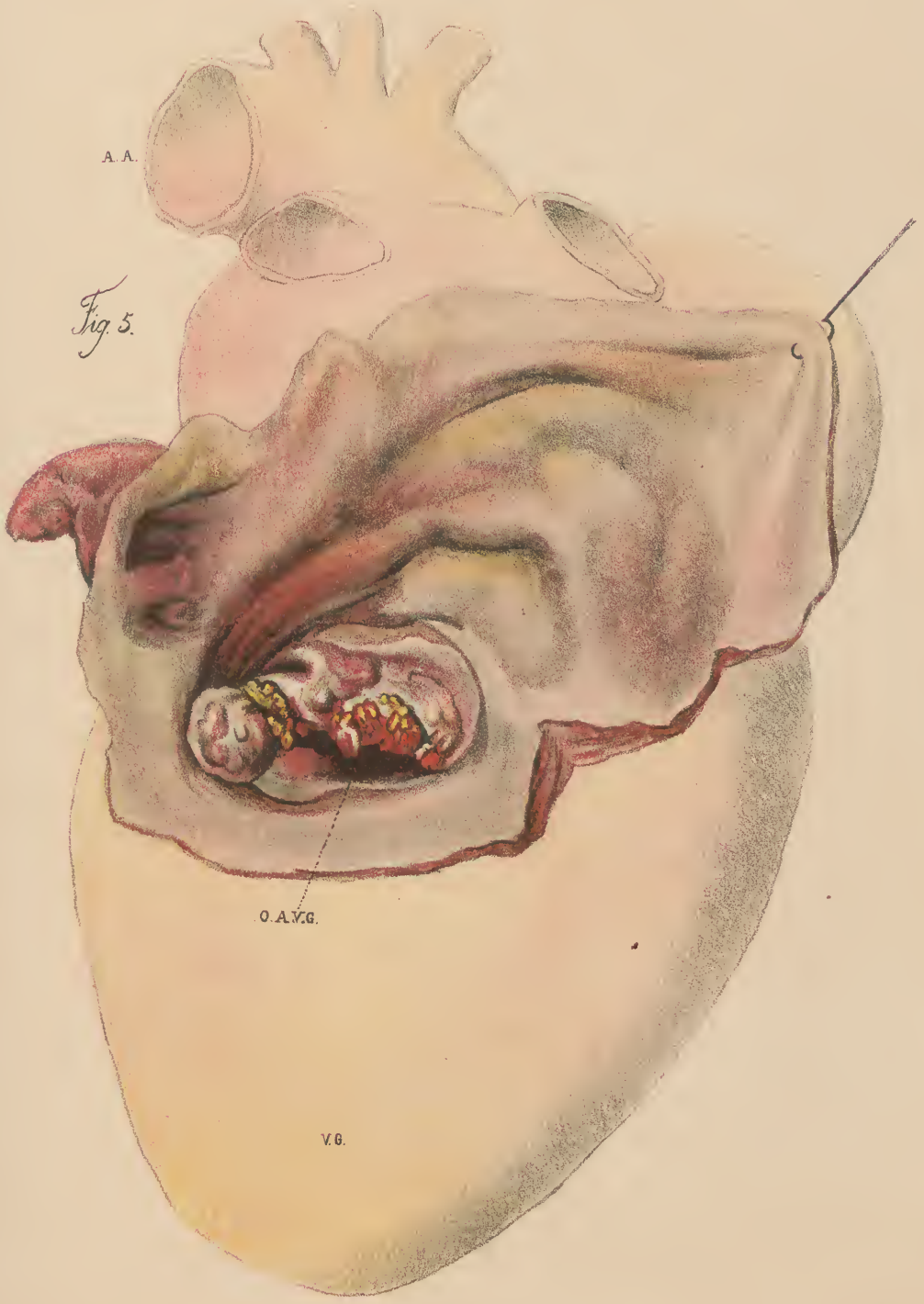
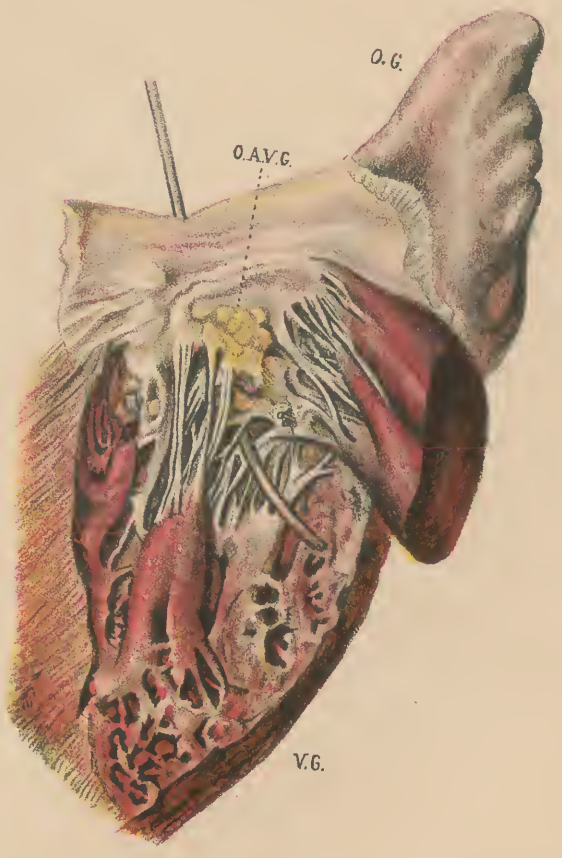


Fig. 6.



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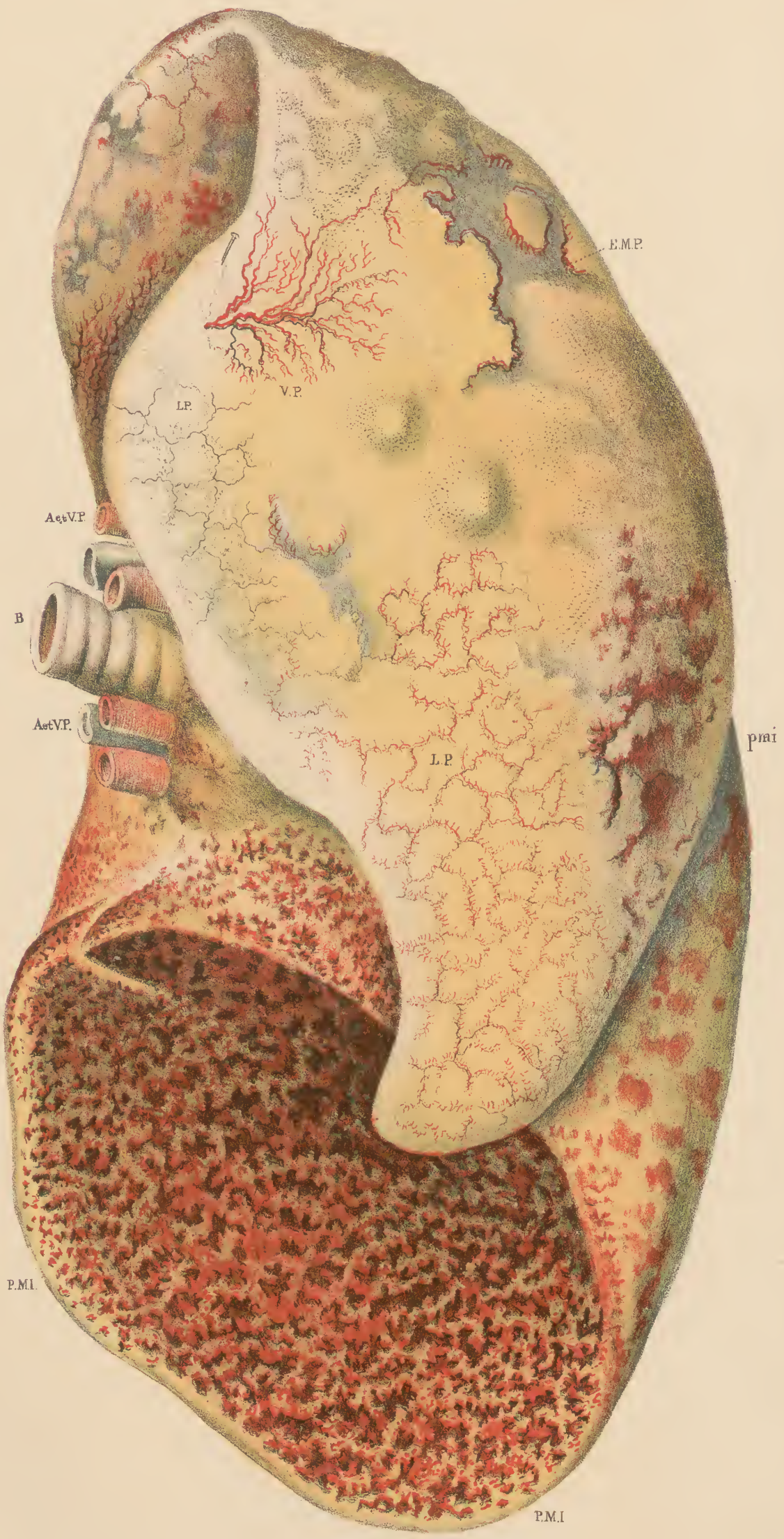
ACUTE CROUPOSE PNEUMONIA.

FIRST AND SECOND STAGES.

Sec. III. *Tab. I.*



DISEASES OF THE ORGANS OF RESPIRATION.

ACUTE CROUPOSE PNEUMONIA.

SECTION III, TABLE I, FIG. 1.

CASE.—Acute croupose pneumonia of the whole of the left lung; with adhesive pleuritis.

Ramett, a law student, 22 years old, was taken sick on the 22d of September, 18—.

Patient had a violent headache about three days previous, and became speedily prostrate. A chill on the 19th; felt some better on the 20th; ate heartily, but soon afterwards felt very uncomfortable; no difficulty of breathing that day; attributed his uncomfortable feeling to indigestion.

21st. Difficulty of breathing and fever; is very prostrate. Attending physician diagnosed inflammation of the left lung.

22d. Livid color of the face, very faint, cannot sit up. Dull sound on percussion over the whole extent of the posterior border, and external surface of the left lung. Bronchial respiration and bronchophony same side. Anterior surface normal. Very thirsty. Sense of suffocation; very thin and very frequent pulse. Temperature 101° Fahr. Excessive shortness of breath. Slight laryngeal cough without expectoration. Evening of same day, shortness of breath slightly diminished.

23, 24, 25, 26, 27th general condition of patient seemingly slightly improved. Urine 1,022 spec. gravity. Large deposit of phosphates readily precipitable by nitrate of silver.

28, 29, 30th. Still further improvement. No shortness of breath. A slight cough, and expectorates some mucus. Dullness on percussion over the left lung posteriorly.

Oct. 1st. Night sweats for the first time the night previous. Expectorates some greenish muco–purulent matter. Skin some hotter than the previous day. Temp. 100° Fahr. Auscultation and percussion same as the day before.

Oct. 4th. Delirium. Tremor of the lips. Color of the face more livid. Lies on the healthy side with ease.

5th. Face purple, hands same color. Lower extremities very cold. Delirious. Very hurried breathing. Pulse 160. Tem. 97° Fahr. Unable to swallow anything. Dies same day.

Post–mortem.—A small quantity of effusion in the pleural cavity. Nearly the whole of the left lung is indurated; very voluminous, and very heavy. Its form is not changed, but looks as if injected, or blown up to its utmost.

Fig. 1 shows a deep depression on its internal surface for the lodgment of the heart. At its base an equally deep depression corresponding with the upper surface of the diaphragm. Fissures of lobes distinctly discernible on the same surface.

The lobules are distinctly outlined on the surface of the lung, and seem to have been each singly affected.

The superior lobe presents on its outer surface a violet color of some of its lobules. It is much depressed at that place, and the softness of its tissue, there, strongly contrasts with the density and toughness of the surrounding lobules (*E.M.P.*).

The purple color was due to severe serous infiltration and stasis of the blood. It has all the characteristics of infiltrated connective tissue.

The whole surface of the affected lung was covered with newly formed vascular networks (*L.P.*), and scattered all over with brownish and blue spots.

These vessels belonged to the neoplastic pseudo-membrane, which was very thin, and could readily be removed. Large vascular trunks (*V.P.*) emerged from below the pleura, the interlobular septa, and from the ramifications of the pleural coverings of the lobules, (visceral portion). The base of the lung was covered with a pseudo-membrane infiltrated with blood (*P.M.I.*).

PATHOLOGY.

MANY years ago, Hasse, in his pathological anatomy of the respiratory organs, correctly observed, that the study of diseases of the organs of respiration has, more than any other, contributed toward the explanation of the nature of inflammation. Since that time the enormously accumulated labors of the best pathological anatomists and pathologists have still further shown that the complex phenomena observed in the process of Inflammation, can be observed in the different forms of Pneumonia with much more ease and greater certainty than in a disease of any other organ. The structural components of those organs consist of epithelial tissue of several forms, large quantities of connective tissue thoroughly interwoven with those; vast networks of blood vessels, in nearly immediate contact with the epithelium of the lungs themselves, with an intervention of only an extremely fine layer of perivascular structure. The singularly thin yet highly elastic cellular epithelium, the existence of which has been denied time and again and repeatedly proven. The sphinctral muscular arrangement in the infudibular entrances, the strong fibrous frame work of the intercellular septa and their relations with the submucous tissue of the bronchii on the one hand, and with the perichondrium of the bronchial cartilages on the other hand. The enormous quantities of nerve structures, fibres, plexuses, ganglia; the glandular structures, both mucous and of other varieties; the abundant network of lymphatic vessels; the large lymphglands and lymphfollicles. All these make it a wonderfully complex anatomical composition. By the side of their anatomical peculiarity is the high importance of their physiological function: "the supply of athmospheric air to the blood and elimination of carbonic acid and vapor of water from it." Notwithstanding the great multiplicity of form observable in the respiratory organs in the graded scale of animal life, the essential elements of their structural arrangement are so simple, that they *consist only of* the *bringing together of a* very fine *membrane* with as thin a layer of blood, and spread out over as wide a surface as possible. The physiological function itself is also based upon one of the plainest laws of nature, that is: the diffusion of gases through a permeable membrane. All these characteristic simplicities and complexities make inflammation of the lungs at once an object of extensive study, and a field for acquisition of clear pathological notions of the essence nature of the process of inflammation.

In order to present a picture of the different varieties of phenomena, manifested in inflammatory diseases of the lungs in so limited a scope as the nature of this work requires, it is necessary to trace its outlines in broad lines. The details can only be filled in here and there. The pathological anatomy must here maintain its first right, though, of course, the clinical side must be as clearly defined. Physical signs, alone readily observable by our senses, indicate the two parallel phenomena of the morbid alterations and morbid manifestations during life. (*Physical symptoms.*)

The change of sound of the thorax over a solidified lung, the rattling, rasping, grating, gurgling noises, discovered in auscultation, only tell us that there is a certain quantity of confined liquid in some cavity; that a movable surface, which ought to be smooth, has become rough and uneven; that a portion of the viscera has become filled with liquid or solid substances in place of the gases, which they ought normally to contain.

What is the nature of the process which brought this about, the simple clinical observations will not reveal. Yet it is of vast moment to the life of the patient to know how it was brought about, and how it is to end; and, especially, what can be done to change its condition.

Croupose pneumonia furnishes the best opportunity of illustrating how a histological process, the several phases of which necessarily succeed each other, can explain the visible alteration of the organic tissue, the modification of its function and the perceptible physical symptoms resulting therefrom.

Step by step each phase of morbid manifestation can be traced.

First Stage.—The vessels of either a whole lobe, or a large por

SECTION III, TABLE II, Fig. I.

A section of the superior and inferior lobes of the lung of the same patient. It shows a whitish, creamy color, and is profusely spread all over with brownish and yellowish white spots (granules). The vesicles show a number of granules, which are made up of blood corpuscles, both red and white, inclosed within a net-work of fine and coarse fibres. "These fibres fill up the vesicular cavity, and are attached to its walls, and communicate with the interalveolar connective tissue." (*Thierfelder's Atlas of pathological histology.*) Between some of the lobules the connective tissue was so soft as to readily allow the lobules to separate, and showed that some of them were affected and some were not.

The whole inferior lobe showed granite-like line markings, and an intermixture of red and cream-colored spots (like some species of marble). In the apex of the left lung a number of small cavities (cavernæ,) freely communicated with the bronchi (*Br.*), and close to it a few scattered obsolete tubercles were found. These were evidently of long standing, and had nothing to do with the present affection. The cavities seemed to have been partly contracted by adhesive cicatrization, and seemed, also, not to have been involved in this present pneumonia.

N. B.—(L. B. on the tables indicate the bronchial glands, which were here very turgid and filled with blood clots. B. bronchi. A.V.& P. pulmonary arteries and veins. L. P. pulmonic lobule).

Figures 2 and 3 show portions of a lung in the second stage of croupose inflammation. Hæmorrhagic infiltration to an extent to completely obliterate all traces of structure. It forms a uniform mass of brick-red tissue, with only here and there the whitish or yellowish outlines of mucus membrane of the bronchi, which have undergone a complete change, from the anæmic condition existing in the circulation of their tissue proper.

These spots resemble, in their appearance, the diphtheritic patches of the larynx and trachea. They are, likely, of the same nature, and seem due to the same cause.

Fig. 4 shows a portion of a lung (of a young man who died of pneumonia). The apex of the right lung had swiftly become compressed by serous effusion, and the structures had become œdematous and soft, showing only here and there distinct traces of the pulmonic tissue.

tion of the lung are turgid, with blood. They are dilated and the capillaries very tortuous and project far into the alveolar cavities, and thereby partly occlude them.

The exudation and the extravasation begin by an infiltration of a rich, albuminous, sticky liquid into the tissues. In the septa and the interlobular pleural structure points of extravasated blood are already visible. The stagnant blood in the altered capillaries permit great quantities of plasma to ooze out through the wall; the power of absorption is for a time perfectly destroyed; the colorless corpuscles along the wall leave the vessels, and the red corpuscles follow soon. This introduces the second stage.

The quantity of exudation corresponds with the intensity of the inflammation.

In intense inflammation, when the circulation in the capillaries has completely ceased, and the current in the veins is hemmed, the hyperæmia drives so much more exudate into the tissue the stronger the forward movement of the arterial blood is toward the capillaries. With the succeeding separation of the colored and colorless corpuscles, the approaching and cohesion of these to the vascular wall, the increased hyperæmia intensifies this stage and enormous exudative masses enter the tissues. In a less intense condition, the hyperæmia being less, the alteration of the vascular wall less, the exudation lighter and the stagnation of the blood in the capillaries easier overcome, the second stage is also very much mitigated, and the following phases pass almost unnoticed.

To the naked eye such a lung is remarkable for its red color (differing in intensity from the brightest scarlet to the deepest brownish-red). Its elasticity is nearly gone; it is tough, dense and heavier than a lung filled with air.

What little air is still existing in the cells can be readily moved backward and forward, it cannot pass the infundibular aperture, for it is occluded by a glutinous substance, which prevents the ingress and egress of air. Dyspnœa is caused by it; equally so the vesicular crepitation. Of course, the lung has temporarily lost the power of respiration. As the patient has as yet not become used to vicariate the healthy lung for the diseased, he will now suffer much more from dyspnœa than afterward.

The lung in Table I, Fig. 1, in this section especially presents the first and second stages of an intense case of croupose pneumonia. The highly swollen, or rather bloated, appearance of the whole organ, the clear definition of the interlobular spaces, the inflamed, injected, and vascularized pleura show that a portion of the lung has already reached the second stage. (We find in the same lung, frequently, several stages of the inflammation, showing this progressive movement of the inflammation from one portion to the other, of the organ.)

Characteristic of the second stage is the enormously injected pleural covering on the lung, on the basal surface, facing the diaphragm. For the mechanical friction, which that muscle exerts upon the surface of the lung naturally causes an increase of circulation in that part of it; and in any inflamed condition it will be found that the hyperæmia there, is greater than in less mechanically irritated portions of the organ.

Red-Hepatisation—In consequence of the alteration of the blood vessels and the stratification of the blood within them, the free surfaces of the cavities of the air cells become covered with enormous quantities of fibrous exudate. At the same time there is an abundant exudation of the colorless, and some colored, corpuscles. All form a complex mass of coarse and fine fibrous threads, which extend from septum to septum, through the infundibular and alveolar cavities, and thus fill them all with the plastic substance. Of course, the air is nearly all driven out of the cells, and the blood in the capillaries, the small veinlets and arterioles, are only motionless clots. "Here and there some vascular cavities are still open, and the circulation in the lung is preserved in a vicarious manner, by increased pressure, resulting from the stagnant condition of the blood in the occluded vessels." (*L. Lichtheim. Die Stœrungen des Lungenkreislaufs, Page 65.*)

A hardened section of a portion of hepatised lung in this stage shows that the vesicular wall is perfectly undisturbed. In the cellular cavity will be seen a finely fibred coagulum, containing large quantities of colorless, and colored corpuscles, derived from the dilated vessels.

The quantity of corpuscular elements, especially the colored, in the coagulum determines the gravity of this stage, and as a general rule, also, the probable termination of the disease-process. When the quantity of red corpuscles is very great, and amounting to severe extravasation, the result is commonly very unfavorable to the patient: for not only does such a condition indicate a deep vascular disturbance, which does not readily recover, but also the blood, as a very easily decomposable substance, will become an additional source of danger to the weakened lung, by inducing secondary disturbances by its decay, etc.

These colored blood globules stamp this stage with the name indicated above. The expectorate, if any exists at all, is dark brownish-red (*rusty sputa*). The color proper to a portion of a a lung in this stage is that of an ox liver, when exposed to the air soon after the death of the animal. It may vary in color, from an intense blood-red to a deep coffee-brown, with reddish specks here and there. The color is not due to the contents of the vessels, but to that of the tissue of the lung. For the quantity of blood in the capillaries diminishes in inverse ratio to the quantity of exudate. The red hepatised tissue becomes heavy and dense.

A cut surface will present a vast number of knotty projections of a tolerably consistent nature. They were once considered to be granulations, but they are really the fibrinous clots in the alveola and infundibula. By gently pressing a cut portion of a lung, soon after its removal from the body, these coagula, or rather fibrinous cylinders, may be pressed out, and will be found, under the microscope, to be casts of the finer bronchi and infundibula.

Remak was the first to correctly interpret their nature, and described them already in 1845: "The bronchial coagula form branched cylinders, dichotomosly divided, the branches being of lesser diameter than the trunk. The main trunk is generally finer than the first embranchments, and ends at its free extremity in fine threads. Very frequently, at the ends of the branchlets, a widened portion is found, etc. . . . . In some of the knotty ends air globules are found, which make them float on water. (Air enclosed by the alveolar clot.) The cylinders and globules containing no air, do not float, but sink to the bottom. When these clots are washed with much water, they are white, and have all the characteristics of washed fibrin. Here and there some spots of blood are found upon their surface." He found them in consumptive lungs, and in cases of croupose pneumonia. In croupose pneumonia of old people, these cylinders contain much black or brown pigment, derived from the lung tissue, and are thrown out with the sputa.

Through this leather-like parenchyma, the inspiratory murmur is continued in the same form as at the entrance of air into the

ACUTE CROUPOSE PNEUMONIA.

'SECOND AND THIRD STACES.

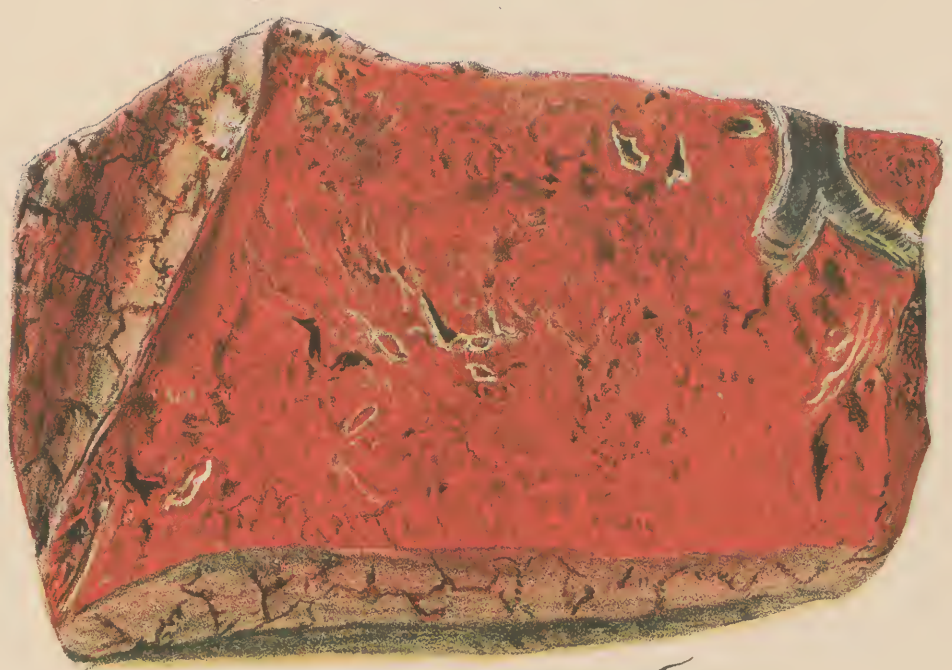


Fig. 2.

Fig. 3.

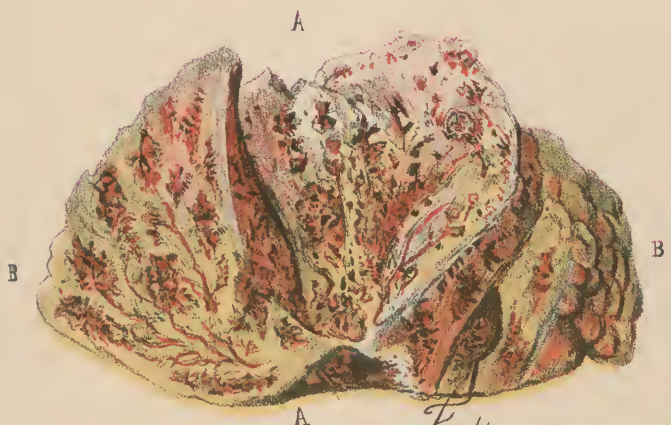
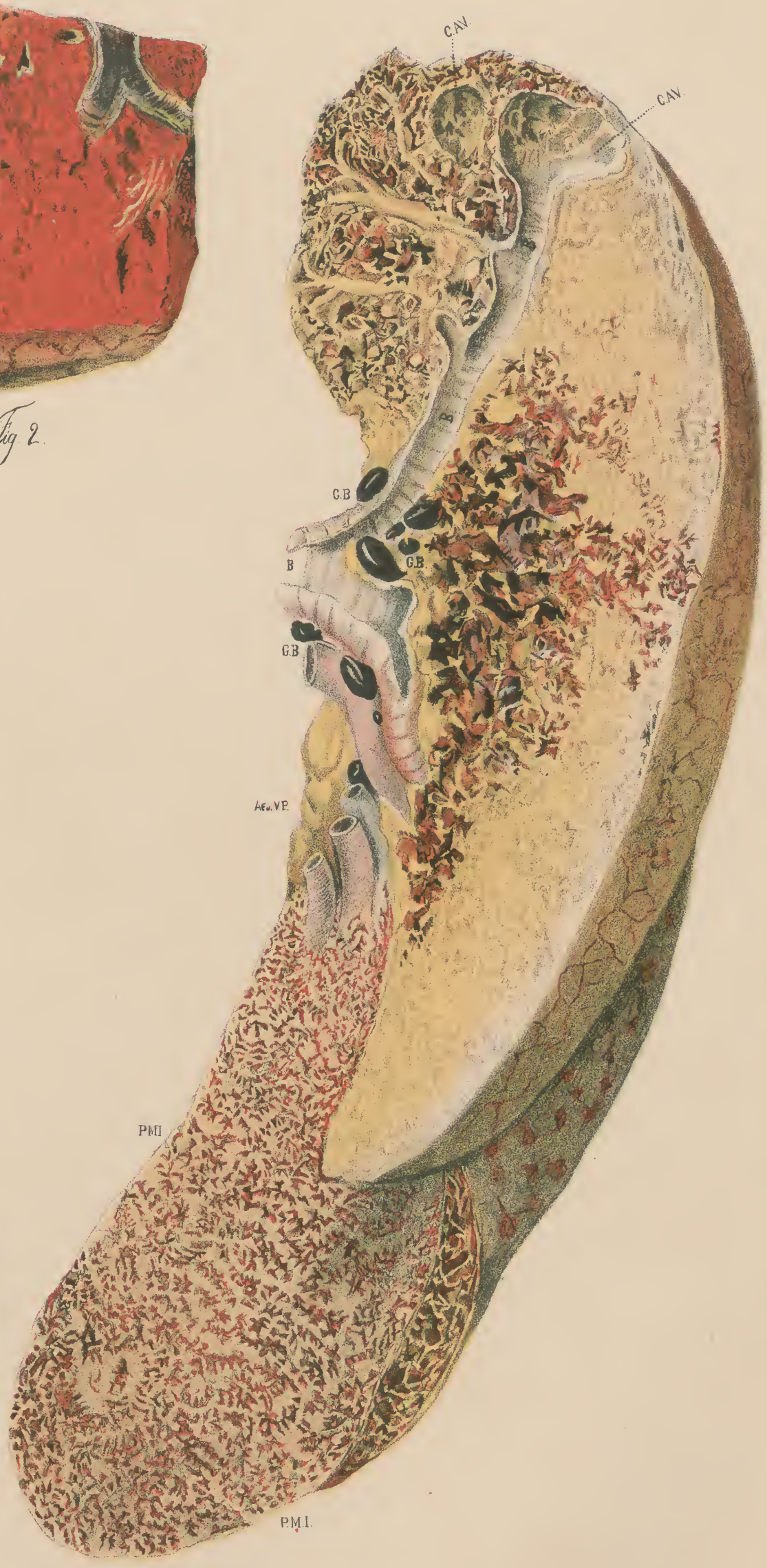


Fig. 4.

Fig. 1.



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ACUTE CROUPOSE PNEUMONIA.

THIRD AND FOURTH STAGES.

Sec. III. *Tab. III.*

Fig. 3.

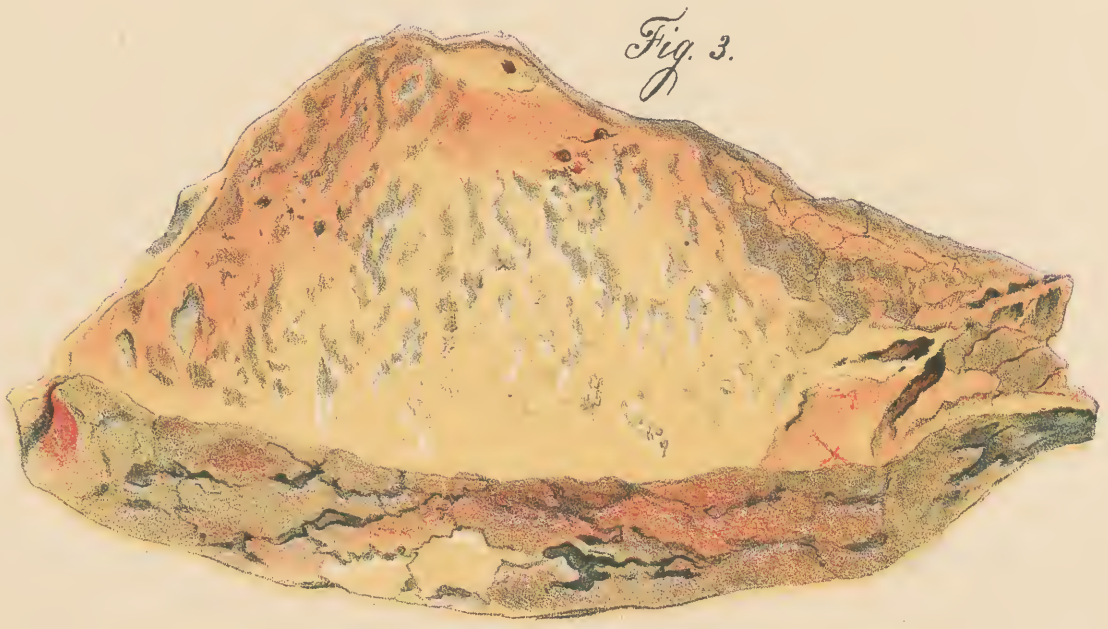
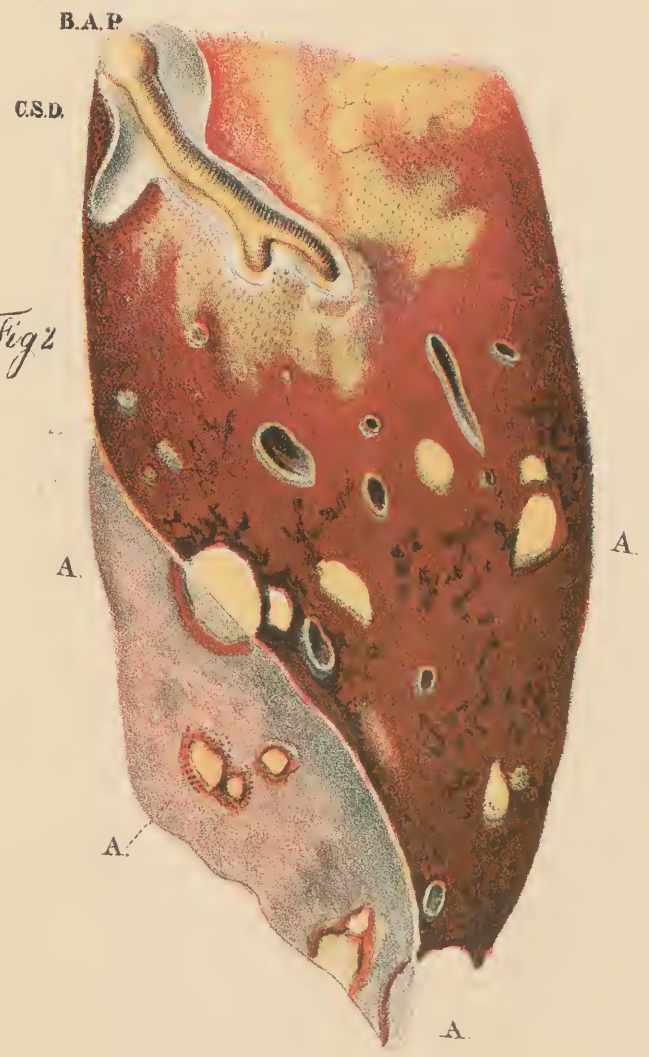


Fig. 1

Fig. 2



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SECTION III, TABLE III.

FIG. 1.—*Asthenic pneumonia, with consequent phlebitis of the veins of the spleen and liver. Metastatic abscesses in the lung, liver and spleen, from Osteomyelitis of the femur, tibia, and fibula.*

NILES, aged thirty years, was wounded by a rifle ball in the knee joint. It broke the tibia and shivered the lower portion of the femur. Amputation was immediately resorted to. The stump seemed to heal very well for some twelve days. There was scarcely any fever. On the thirteenth day he was taken with a severe chill, followed by a strong fever and very profuse perspiration. The wound became suddenly dry and covered with a greenish gray membrane (dry gangrene).

The muscles of the thigh became contracted. The patient experienced no pain in the thorax, nor in the abdomen, even on strong pressure. The pulse gradually diminished in volume and force. General loss of bodily energy and strength. Dies under very severe symptoms on the 18th day after the first chill.

Post-mortem.— The spleen is found much enlarged. Scattered in its structure were found many red spherical masses. (Hæmorrhagic focuses). Incipient phlebitis of the splenic veins.

The liver presented many slate-colored spots in its parenchyma. These were found, on incision, to be masses of concrete pus, with some degenerated liver tissue. Both mingled, and presented a marbled appearance. These were surrounded by slate colored coverings and had the appearance of being in the first stage of abscess of the glandular structure of the liver. In the lung there existed purulent focuses (*Metastatic Abscesses*).

Fig. 1 presents the right lung. At the base (*B.*) and the posterior border of the inferior lobe (*B. P.*) the abscesses (*A. A. A.*), which were small and irregular, projected from the surface of the lung: they were surrounded by an indurated red circle. The superior lobe is completely free from any disease.

Fig. 2 shows that the abscesses are not all superficial, but that some of them occupy the internal portion of the organ. The pulmonary artery contains some very thick clots (*C.S.D.*), which are very adherent to the vascular wall (*B.A.P.*).

The marrow of the stump of the femur, and the spongy tissue of the head of that bone, were all filled with pus.

Fig. 3 presents a portion of the lung that had undergone gray, or slate-colored, and yellow or purulent hepatization.

trachea and into the bronchi without changing its tone to the ear of the auscultator. Bronchial respiration will be heard mingled with some coarse vesicular rale, but without adventitious vesicular or fine crepitating rhonchus.

Percussion sound over the affected lung is perfectly dull, and the affected portion can distinctly be marked.

Tables I and II of this section render a very correct representation of the various phases of the croupose inflammatory process, and show several degrees of gravity of the second stage.

Third Stage. Yellow Hepatization.

This stage is chiefly characterized by the activity which the lung tissue, which was hitherto in a passive state, now displays.

The epithelial and connective tissues of the alveolar parenchyma now react by a process of highly active cell-proliferation. In every interstice of the vessels large quantities of new cells are formed.

The whole surface is covered with a many layered epithelial element mingled with great numbers of lymphoid bodies. This is a genuine condition of catarrhal inflammation. The increased cellular elements render the affected lung still heavier, and more voluminous; its density and toughness also increase. The little fibrinous coagula are now not so prominent. Most remarkable is now the change of its color. The red is turned into yellow or yellowish white, cream color. This is due to a still further diminution of the vascular contents, produced by the enormous pressure exerted upon them by the swollen tissue. The blood corpuscles and fibrinous tissue gradually become discolored, and the purulent elements become more prominent.

Even in this stage some blood-vessels are still kept open and their blood in motion, by the increased pressure upon the unaffected branches of the pulmonary artery. In this stage there is a gradual weakening of the whole heart. The right ventricle has struggled hard to maintain the circulation of the pulmonary artery, in branches still free from inflammation by an over-pressure, and the left, in order to keep up the nutrition of the heart, and form a temporary hypertrophy of the right ventricle and maintain the elasticity of the arteries. The force of the heart enters here as the great arbitrator of the fate of the patient.

The danger to the life of the patient in the second stage is blood-poisoning and anæmia of the brain. The great mass of the blood is not sufficiently aerated, for in those vessels where it can still flow, it passes with such swiftness that it has not the necessary time for the exchange of gases. In the stagnant vessels there is barely any diffusion at all. A deep state of stupor and yellow tinge of the skin indicates a lack of arterialization of the blood.

As the croupose pneumonia affects mostly the lower lobes, the swollen portion moves backwards and ascends sometime above the third rib posteriorly, (see Tab. I, Fig. 1,) whilst in front it barely touches the thorax. The middle and the upper half of the superior lobe occupy nearly the whole anterior thoracic surface, whilst laterally it is covered by the inferior and half of the superior lobes.

In the third stage the lung has lost very much of its toughness and dryness; on the contrary, the affected portions commence to yield to an impress of the finger; and if a large part of a lobe or a whole lobe is affected, distinct imprints of the ribs, against which the swollen lung was pressed, will be visible.

The fourth or purulent stage, or resolution, begins by a gradual or sometimes sudden metamorphosis of the exudation.

The cell production of the third stage gradually diminishes, the fibrinous strings attached to the alveolar wall loosen, and melt into a jelly-like mass, which encloses all sorts of corpuscular elements, forming a little lump in each air-cell. Both the corpuscular elements of the exudate, as well as those of parenchymatous origin, can be found in the sputa at that stage. Generally it is mixed with some broken-down blood corpuscles and blood pigment.

Treated with acetic acid, it will cause a precipitate of *mucin*, showing a change of fibrin into mucin. During the red hepatization the sputa, treated with acetic acid, will clear the exudate and dissolve the fibrin. Undoubtedly a very notable quantity of the mucin is derived also from the epithelial cells of the bronchial and alveolar epithelium.

Portions of an affected lung are very slippery and slimy, and can be readily crushed between the thumb and finger, and indicate a condition of great softening.

The grayish-white anæmic substance contains no fibrinous granules as before, but a considerable quantity of concrete and soft pus. This whole stage is a conversion of the exudate into a readily soluble substance, which is generally expectorated by the lungs. The least portion of this mass is normally absorbed or converted into fat and removed by the circulation. The disagreeable cough produced by these now excrementitious substances, which cannot be readily removed by the bronchi, partly on account of the still contracted sphinctral entrances of the infundibula, and partly from insufficient elasticity of the parenchyma, is very harassing to the patient, and causes sleeplessness and great uneasiness, and continues until the whole mass has become liquified and readily removed.

The defervescency in the third and fourth stages generally increases and constitutes longer remissions in the fever. Toward the end of the fourth stage healthy individuals commence to feel notably improved, and with the gradual cessation of anæmia (in measure as the exudate is removed) the appetite and system gain in proportion. The circulation is often established in the relaxed lung tissue in an overwhelming manner, and there are sometimes produced ruptures of that tissue, with extravasations into the pleural cavity. These are, as a rule, soon reabsorbed.

This lack of elasticity of the lung tissue persists in some cases for a long time, and keeps the patient in a very uneasy condition.

PHYSICAL OR OBJECTIVE SYMPTOMS IN ACUTE CROUPOSE PNEUMONIA.

The inflammation of the pulmonic parenchyma produces several alterations in the phenomena of sound, discoverable by auscultation and percussion.

The catarrhal condition, always present to a greater or lesser extent in pneumonia, also the modified respiratory processes taking place, determine the altered phonetic manifestations, during the several stages of the inflammation.

Sound on Percussion in the First Stage.

As long as the pulmonic parenchyma is not filled with exudation, and its contractility is not altered, the sound will not differ from the normal, however much the vessels may be filled. Only

SECTION III TABLE IV.

Fig. 1. (Lobular pneumonia following phlebitis of the pulmonary veins). Mary A. D., twenty-nine years old; primapara, confined July 12, 18—. Parturition of short duration; artificial delivery. Considerable hæmorrhage subsequently.

Symptoms.—Face pale, small, frequent pulse, complains of being very feeble; excessive pain in contracted uterus, and increases on the slightest pressure. Patient in a high state of anæmia.

13th. Hypogastric pain slightly diminished. Less fever.

14th. Flow of milk into the mammae.

15, 16, 17th. Excessive perspiration, great thirst; very nervous; headache; inclined to faint. Abdomen not painful.

18, 19, 20th. Appears considerably better, but the pulse continues all the time very frequent. 21st. A return of the hypogastric and abdominal pain. Some diarrhœa; some relief.

23d. Severe pain under the false ribs. Exploration of thorax does not reveal any pulmonic trouble.

25th July to 3d Aug., improves gradually. 4th. Suddenly, severe dyspnœa; cough without expectoration; exceedingly frequent pulse; very nervous and oppressed. No pain in the hypogastrium.

Thoracic exploration yields little result. Normal sound on percussion. Auscultation reveals a slight gurgling tone at the base of the lung behind. Secretion of milk suddenly checked.

5th Aug. Face looks changed, expression very anxious. Very small and frequent pulse; dry tormenting cough. Some diarrhœa produced by a cathartic.

6th Aug. Has no pain. Respiration very hurried. No expectoration.

9th. Dies under symptoms of asphyxia.

Post-mortem Autopsy.—No trace of peritonitis; no apparent lesion of large or small intestine; stomach sound. Uterus slightly enlarged. Uterine, ovarian and hypogastric veins like whipcords, and full of compact adherent and colorless clots. The external iliac artery, the left crural artery and some of their branches, are also distended by the same kind of clots, but not to the extent as the veins.

At the base of the left lung (Fig. 2), some small, whitish-green purulent focuses are to be seen (*A. L A. L.*) They are superficial, and are distinctly visible through the transparent visceral pleura. In the anterior portion of one of these, there is a small adherent eschar. Several other points of the surface of the lung are of a variegated reddish color, and seem rather compact. One of these points (*F M*) looks like an apoplectic focus.

The posterior half of the inferior lobes of both lungs are perfectly infiltrated with serum and pus, and are unfit for respiration.

Incisions here and there into the lungs showed hard concretions filling up the branches of the pulmonary artery (*A.P.*) A careful dissection of this vessel revealed colorless bloody clots, slightly adhering to the vascular walls. These clots could be followed into the finest branchlets of the artery.

It could readily be seen that the embolic occlusion began in the trunks, and spread into the branches. The clots in the smaller vessels were red, whilst in the larger and in the trunks they were colorless.

Within these clots a quantity of pus was found. In (*C. P.*) the clots are colorless, but contain no pus.

Fig. 2 represents a transverse section of the same lung. There is to be seen the irregular, spheroidal shaped inflammatory focuses of the lung (*A.L. A.L. A.L.*), which have not yet reached the stage of formation of abscesses. In nearly all of these, a thick, red layer of indurated tissue surrounds a whitish, hard mass composed of pus, infiltrated into the pulmonary parenchyma.

Here and there reddish points of the indurated structure are perceptible in the midst of the infiltrated pus.

Some of the focuses present a commencement of collection of pus, and the vascular tufts, which traverse the walls of those focal infiltrations, can readily be made visible by the use of small forceps drawing them forward from their position. The infiltrated blood had completely covered the parenchymatous structures without, however, destroying them.

The other lung, with the exception of the pleuritis, presented the same anatomical alterations.

Such appearances seem distinctly to indicate that the pus must have formed locally during, and by, the inflammatory process. (*F. M. Pseudo-membrane*).

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with the infiltrated condition of the lung tissue, or the loss of elasticity will the percussion sound become modified. The extent of modification will be in accordance with the relative quantity of air to that of the exudate in the organ. The intensity of the inflammation will not determine it.

The infiltrated portion will yield more of a tympanitic sound so long as it contains some air; provided the thoracic wall be not too resistant. This tympanitic sound is somewhat muffled, and remains for some time in this condition until the infiltration fully takes place, and then the sound becomes dull—a peculiarity which will indicate that hepatization has commenced. This is due to the loss of elasticity of the lung-tissue. This tympanitic character is sometimes very persistent, and sometimes speedily disappears, and yields to the dull sound. Before such a sound appears the lung must be infiltrated to the thickness of at least an inch.

The adjacent portion of the lung which is not infiltrated yields a normally clear sound.

Sound—Phenomena on Auscultation.

Simple vascular hyperæmia, without infiltration into the tissue, and without accumulation of secretion in the air passages, does not alter the auscultatory phenomena. As long as the air passages and the parenchyma are still accessible to air, though there be infiltration in the one and accumulation of secreta in the other, there will be no other sound manifested than such as is heard in bronchial catarrh, with air passages filled by liquid secretion. The kind of Rale depends (both at the beginning of the inflammation and at the stage of resolution) on whether the liquid is contained in the alveola and finer bronchi alone, in the larger bronchi too, or only in the larger bronchi; whether the infiltrate is very tough; and whether the respiration is accelerated to a greater or lesser extent.

In the exudative and in the resolvent stages of pneumonia, all sorts of rattling, humming, whistling and seething sounds, or rather noises (misnamed murmurs), may be heard singly or conjointly. They are generally audible not only over the inflamed portion, but also beyond it; and often over the whole thorax. These abnormal noises (*Bruits*) often cover the respiratory murmur—both vesicular and indefinite. They are as often—especially when respiration is carried on slowly—heard simultaneously with the normal. Very quiet breathing generally dissipates the abnormal *Bruits*.

Stage of Hepatization—Percussion.

Provided the thickness of the hepatized part of the lung, which lies against the thoracic wall, is at least an inch thick, and its extent at least three inches in diameter, the sound emitted by it will be muffled, and the resistance of that portion of the thorax will be greater than usual. The sound becomes duller and the percussing finger meets with so much more resistance, the greater the thickness the hepatized portion and the more extensive its area.

The flexibility of the thoracic wall must be taken into account, to exactly ascertain the degree and extent of the hepatization by percussion.

The more muffled the sound the greater is the depth of the hepatized portion. When the sound is absolutely dull, even below the most flexible portion of the chest, it may safely be assumed that the whole depth of the lung is involved in hepatization. The adjacent portions of the hepatized part may either be infiltrated, but still containing some air, or *free* and *normally* distended, or *abnormally* distended, that is, emphysematous.

The latter condition exists most frequently near the border of the lobes. Parts remote from the hepatized portions may be normal; emphysematous, infiltrated with serum, or with more consistent liquid, but still containing some air. An emphysematous portion of the lung bordering on a hepatized portion will emit a clear tympanitic sound; whilst an emphysematous part distant from the hepatized will have a somewhat more resonant, but no more tympanitic ring than a normal lung. Infiltrated but still air containing portions, situated closely to the thoracic wall, emit also a more tympanitic sound than a normal part.

It is, under all circumstances, best to percuss the corresponding

ACUTE LOBULAR PNEUMONIA.

Fig. 1.

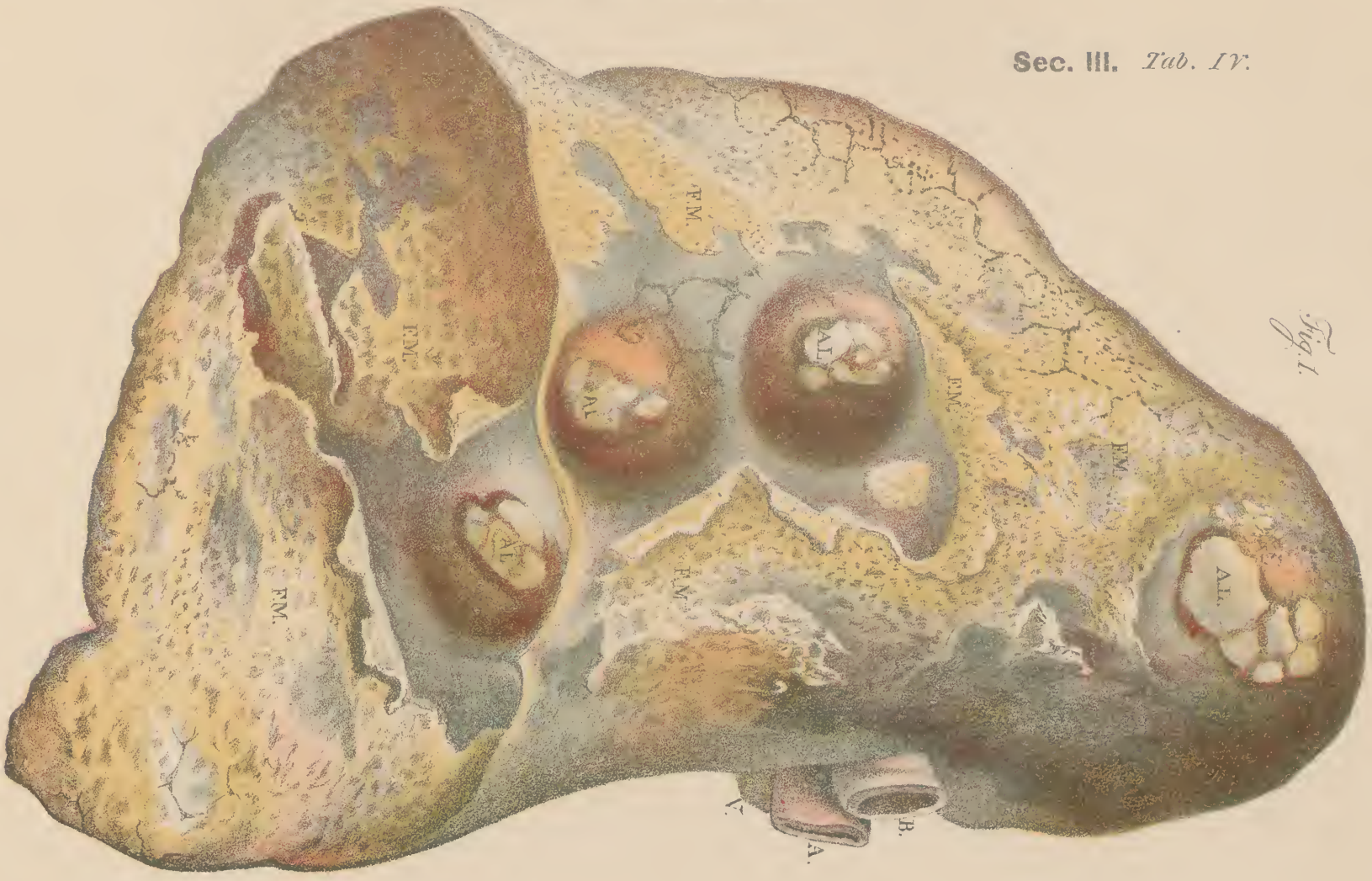
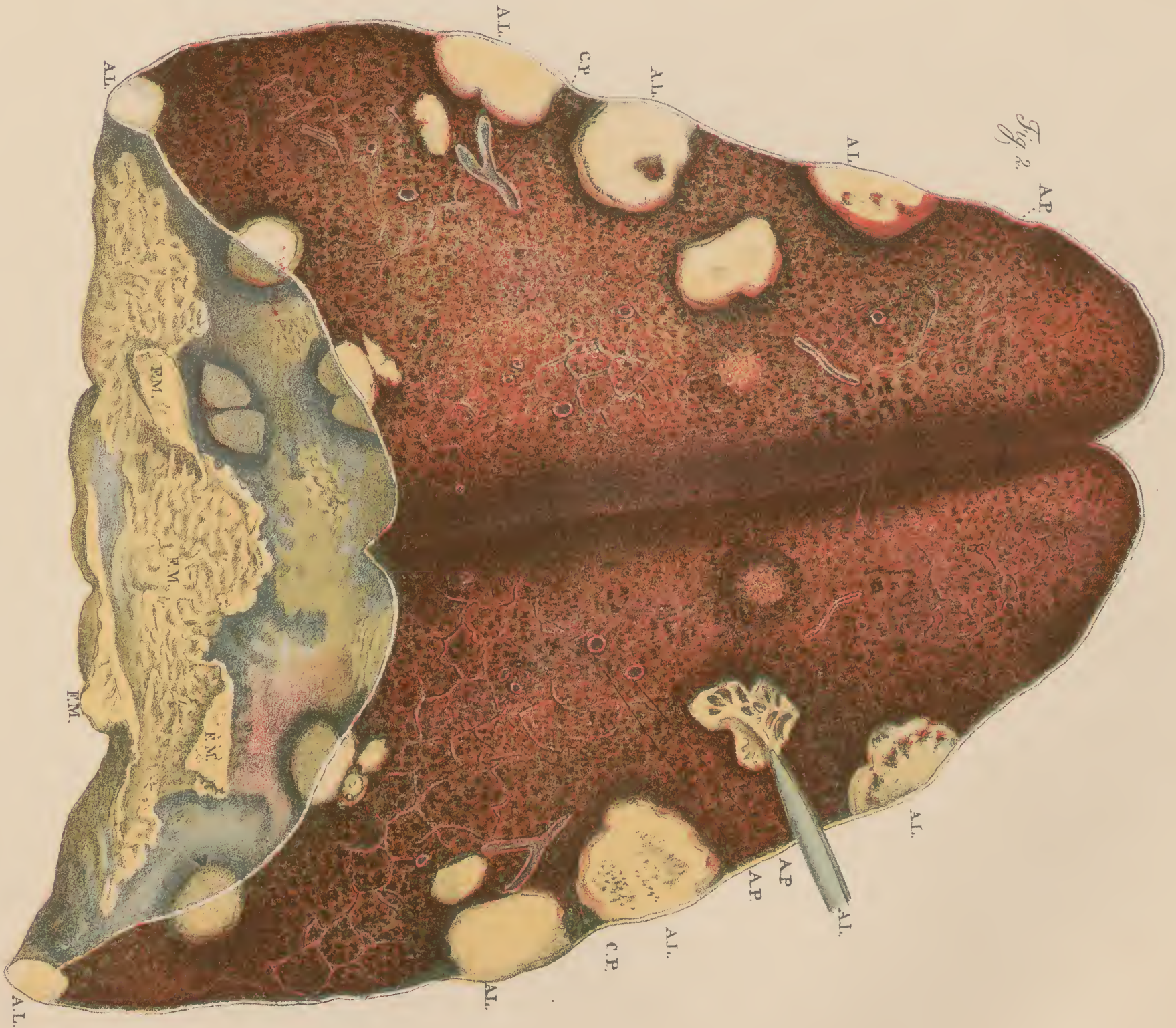


Fig. 2.



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Sec. III. *Tab. V.*

CANCRENA PULMONUM.

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TABLE V.

DIFFUSE GANGRENE OF THE RIGHT LUNG.

CASE:—*G. F.* A man of 38 years.

History and Symptoms.—Entered hospital Feb. 24, 1874. Had been sick about nine months, had a severe cough, copious expectoration, and suffered from dyspnœa and hæmoptysis. About a month ago he contracted acute pneumonia. Present symptoms: Percussion, dull sound posteriorly over the whole of the right lung. Auscultation: bronchophony, with signs of a large cavity about the middle of the right scapula. The sputa very copious, of a grayish-green color, and so fetid that he had to be isolated, notwithstanding all deodorants used about his person. He died of colliquative diarrhœa and exhaustion two days later.

Post mortem.—There was found a large gangrenous cavity in the right lung, occupying the lower portion of the upper lobe, and the upper portion of the right lower lobe. The cavity contained about six ounces of blackish, highly fetid serum. The middle and lower portion of the same lower lobe was in a state of gray hepatization. The left lung was congested, and showed a small cavity in the apex. Both lungs were filled with great quantities of miliary tubercles.

Figs. 1 and 2 show the same lung. Fig. 1 shows the lung covered by the pleura (*P.*), portions of which are perforated (*P.P.*); beneath the pleural covering the dark color of the gangrenous tissue is slightly visible. Fig. 2 shows the pleura removed, the cavity exposed; (*F.G.*) gangrenous excavations, and sphacelous tissue; (*E.*) exudate between the pleuræ; (*P.P.*) perforations; (*P.*) pleuracostalis which is adherent to the visceral plate; (*B.A.*) anterior border; (*B.*) border; (*B.P.*) posterior border.

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portions of the healthy lung, and even several parts of the diseased, to fully ascertain the exact peculiarity and depth of the sounds of healthy portions, to better compare them with those of the diseased parts.

Sound—Phenomena by Auscultation.

When the hepatized part is at least of an extent sufficient to involve one of the larger branches of the bronchus; farther, when the bronchial branch is neither filled with liquid, solid exudate, or blood *coagula*, and of course filled with air, which freely communicates with the trachea, then the voice of the patient will consonate with the bronchus and will become audible in the portion of the thorax nearest to it, to a greater or lesser extent. (There will be bronchophony.)

In this stage there will be heard either the sound of bronchial respiration alone, consonating, rattling, whistling, sibilating, humming noises with the bronchial respiratory sound, single abnormal *bruits*, or several at the same time. All will be the more audible, the deeper and the more rapid the respiratory action will be carried on by the patient. It does not necessarily follow that *bronchial* respiration should always co-exist with bronchophony. For very often, even with the most pronounced bronchophony, there is heard only an indefinite respiratory murmur,—though never vesicular murmur,—on the other hand, bronchial murmur alone, or with all sorts of abnormal noises, may exist without simultaneous bronchophony.

In case the hepatized part is confined to a lesser space than that mentioned above, or when the bronchial branch is filled with an exudate, which excludes the air altogether, then no consonance takes place in the hepatization. Neither bronchophony, bronchial respiratory murmur, etc., become audible. The voice of the patient is either unheard over the hepatized part or perceived as an indefinite murmur, and then very muffled. All other *bruits* are either not at all or but very indistinctly heard. All the above described sounds may suddenly become audible, and for a time continue to be heard, when a sufficient quantity of expectoration has freed the air passages either by coughing or otherwise.

The described sound-phenomena exist in the red, yellow, or gray hepatizations. Even abscesses formed during those stages will not modify the nature of the sounds.

Clinically different, although anatomically identical with the above described croupose pneumonia, are several forms of the disease, which were formally classified as typhoid pneumonia, bilious pneumonia, but have, at present, received the generic name of *asthenic* pneumonia. They all have one character in common, that of asthenia, but differ from each other by a number of groups of symptoms. *Leichtenstern* has, in a masterly manner, discussed the subject-nature of this asthenia in the 82d *lecture of Volkmann's collection* of clinical lectures. He differentiates primary from secondary, or idiosyncratic. The last befalls very old people, cachectic and anæmic individuals, drunkards and very dissipated persons. In fact, in all of whom there exists great weakness of the muscular tissue of the heart and general *atony*.

Primary asthenic pneumonia is characterized:

1. By prodromal symptoms during several days, prior to the manifest attack, resembling those of infectious diseases.
2. As a rule, the attack begins without a chill, or much sensation of chilliness.
3. Scant infiltration in the lung tissue.
4. Speedy transition from the red into the gray hepatization. Then purulent infiltration, with very ready formation of abscesses and gangrene of the tissues.
5. The upper lobe is the most frequently affected.
6. Very early prostration, delirium and coma (this form was called typhoid).
7. The fever is usually very high.
8. Inflammation and swelling of the liver and the spleen, also albuminurea are very frequent.
9. Icterus and some vomiting (formerly called bilious pneumonia.
10. The disease is most frequent in the summer and fall; very seldom in the spring.
11. The rate of mortality is very great.

The author considers the peculiar malignity of this disease to be due, not so much to the intensity of the disease-process as to that of the infection.

The typical succession of the histological changes in this form of pneumonia exist, but in a very irregular mode of development. Some stages last longer than usual, others pass imperceptibly or are suddenly arrested by necrobiotic processes.

Until very lately, cold and heat, or great and sudden changes of temperature, were considered the chief agents of croupose pneumonia in all its forms; but recent very careful observations and accurate experiments, made upon animals, have shown that neither of those agencies are able to produce a form of inflammation resembling either of the named forms of pneumonia with its typical phases. On the other hand, the belief in the infectious nature of pneumonic origin gains more and more ground.

Literature on the subject of pneumonia is lately teeming with reports and descriptions of many forms of infectious pneumonia, in different parts of the world.

We find, for instance (in *Doerenberg, Berlin, Dissertation*) epidemic pneumonia combined with typhus, malaria, diphtheria. The fever was very high. Very copious haemoptysis. Cerebral symptoms very severe. (*Banti, epidemic pneumonia.*)

Ritter, on epidemic pneumonia with typhoid type. Wiedenmann, Zur Lehre, deutsch, arch. f. clin. medezin. Vol. 25, 589 p., describes microscopic examinations of lung tissue in infectious pneumonia:

"There were thrombotic coagula in the lymph vessels, containing enormous quantities of bacteria. The parenchyma surrounding these vessels was infiltrated with colored and colorless corpuscles. The pulmonary artery and all its branches contained thrombi, which were filled with bacteria; this was also the case with the finer bronchi."

Wynter Blyth describes infectious pneumonia, existing in September, 1875.

Wrigly, Grimshaw and *Moore*, in *Dublin Journal of Med., Aug., 1875*, call that form of epidemic *pythogenic pneumonia*, which they attribute to miasmata or to a zymotic origin.

Besides the normal termination, that is resolution and restoration to health, croupose pneumonia may terminate in tuberculosis or gangrene, according as the infiltrated tissue be more or less anæmic, and the re-establishment of circulation be retarded.

The eminently acute observer *Laenec* was the first to describe gangrene of the lungs, in his *Traite de l'auscultation, Paris, 1831*, and it holds good to this day. He differentiates between a circumscribed and a non-circumscribed form of gangrene and says: "The first constitutes a dry, black, gangrenous eschar, which is distinctly circumscribed. In the second form the tissue is moist, very fragile, of the consistency of the lung tissue in the first stage of inflammation, and varies in color from a whitish-gray to different shades of green. By spots it is perfectly soft and deliquescent. An opac, sanious liquid, exceedingly fetid, oozes from a cut surface. The mortified tissue imperceptibly passes into a zone of inflamed structure, separating it from the healthy portions. The circumscribed form is always confined to small portions of the parenchyma, and is not much inclined to spread, whilst the non-circumscribed may occupy a whole or a part of a lobe. There are three stages in its development: 1. The beginning stage of mortification. 2. The sphacelus. 3. The sloughing and formation of cavities.

The sputa, from a gangrenous lung, are of a greenish or brownish color, or of several shades of gray, always contain more or less *pus* and emit an insupportably fetid odor of gangrene. In the early stages, opac and milky, they turn gradually darker. When the tissues begin to heal, the sputa turn yellowish and have an odor of pus, gradually losing its fetor."

He farther very correctly remarks that pulmonary gangrene does not nearly as often originate in inflammation as is usually believed.

Dietrich in his work "*On Gangrene of the Lungs, 1850,*" and *Traube* in his classic "*Collection of Researches,*" have both clearly demonstrated that by far the greatest number of cases of pulmonic gangrene are due to bronchial dilatations, when the putrid bronchial secretion stagnates in the finer cavities of the air passages and undergoes metamorphosis consisting of either calcification, inspissation or liquification. The latter is thrown out by expectoration as a *very putrid sputum.* This sputum serves as a very valuable diagnostic agent. *Traube* has microscopically examined it and found it derived from two distinct sources, one from what he termed *putrid bronchitis*, and the other from genuine mortification and decay of the lung tissue proper. "*The bad odor of the sputum,*" he says, "its dirty yellowish green color, its liquid form, which permits it to separate, after standing for a while in a vessel, into three distinct layers: the upper yellowish green, opaque, foamy; the middle clearly purulent; the lowest yellow, opaque and resembling a sediment of broken and unbroken pus corpuscles; finally the soft fetid lumps which it contains, and which are agglomerations of crystals of the fatty acids, having a smooth shining surface, characterize it as a one from *putrid bronchitis, or pulmonary gangrene.* In that from a gangrenous lung, there are also present particles of destroyed

TABLE VI.

FIG. 1.—*Catarrhal pneumonia, subsequent to uterine, hepatic and splenitic panphlebitis. Thrombotic occlusion of the pulmonary artery and many of its larger branches. Subpleural lung tissue ulcerated in many places. Hœmorrhagic infarct at the base of the left lung.*

CASE.—A woman 28 years of age. Primapara.

History.—Hard labor, artificial delivery, followed by enormous hæmorrhage. Was taken with metritis on the second day after the birth; phlebitis of the uterine veins appeared next day. Two days later, excessive pain in the chest, and severe dyspnœa. Died in a collapse twenty-eight hours after the birth of the child.

Post mortem.—Abdomen, no sign of peritonitis. (There was no microscopic examination of the membrane). Intestines very hyperæmic. Uterine, ovarian and hypogastric veins thrombotic. They are hard and feel like cords. The external iliac, the femoral veins and their branches, also filled with compact and adherent thrombi, but of more recent date; at the base of the left lung several small abscesses filled with pus. They are circumscribed by some anæmic tissue, and are superficially situated—subpleural. Some are yellowish-white, others are red. Their surrounding parenchyma has a mottled appearance, and of several shades of red. At (*F. S.*) there is infarct into the lung tissue. The posterior halves of both lower lobes are perfectly infiltrated with serum and unfit for breathing. Incisions into different parts of the left lung show the pulmonary artery and many of its branches to be filled with thrombi; red in the smaller, colorless in the trunk and larger branches. Within many clots liquid pus was found. (*A. P.*) pulmonary artery, (*C. P.*) collection of pus, (*C. S. D.*) colorless thrombus, (*B.*) bronchus, (*S.*) superior lobe.

Figs. 1, 2, 3. Softened caseous masses; fig. 2, shows commencement of destruction of the parenchyma near a small bronchus; fig. 3, a number of cavities produced by the ulcerative corrosion of liquified caseous matter, (*A*); bronchial tube, (*B*); cavities and ulcers, (*C*). The mucous membrane of the bronchial tubes is very red, and in spots dark, and infiltrated with black pigment.

Fig. 4. Portion of caseous lung near the hilum; (*A.*) caseous change obliterating all the structural peculiarities of the affected lung; no interlobular septa are noticeable in this region.

Fig. 5. Caseous pseudo-tuberculous masses on the outer surface of upper lobe. The interlobular septa are very much thickened and are very distinct, the lobules are very much contracted near the caseous portions; the pseudo-tubercles (solid caseous collections) are spheroidal in shape, and project above the pleural surface. They vary in size; (*A.*) tubercles, (*B.*) bronchial mucous membrane, partly red, partly brown; enlarged and dilated blood vessels run parallel to its long axis.

Fig. 6. Miliary tubercles in a highly congestsd portion of a lung; (*B.*) small bronchial tubes, partly filled with thrombi; (*P.*) pleural surface.

Fig. 7. Portion of upper lobe of a lung infiltrated with caseous masses; the tissue is nearly solid and in a state of brown induration.

Figs. 8, 9. Tubercles in apex of a lung; the parenchyma has lost its lobular arrangement, and presents a nearly homogeneous appearance. Dilated blood vessels and lymphatics cross the tissue in different directions; they are situated outside the visceral pleura, which is firmly united with the lung. A number of small and large grayish-blue tuberculous masses are situated partly sub-pleural, partly on top; they have a gelatinous consistence and are slightly transparent; they typify Laenec's gray tubercle in its primary stage.

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lung parenchyma. These particles are surrounded by grayish yellow masses with fringed edges, and consist of elastic, transparent, colorless substance, and contain very numerous fat molecules and other granular detritus, mingled with groups of black pigment and crystals of fatty acids. Vast numbers of vibrioni are found in the sputa." *Leyden* and *Jaffe* have also discovered great numbers of vibrioni in the gangrenous sputa, as well as in the dilated bronchi themselves. The vibrioni were endowed with very lively motion resembling the amœboid. The microorganisms are similar to those found on the teeth and the gums in putrid stomatitis. Large quantities of crystals and amorphous substances, usually to be met in organic substances undergoing putrefaction, such as leucin, tyrosin, etc., were also found therein.

Occasionally any of the forms of croupous pneumonia may terminate in the formation of pulmonary abscesses. The older pathologists have confounded this mode of termination with the gangrenous, for some really good reason. In nearly every severe form of pneumonia there is mortification of the lung tissue, but to a very limited extent, and the necrotic process passes so quickly into the reparative stages that no gangrenous manifestation becomes apparent.

Whilst formerly pulmonary abscess was thought to be a very common termination of fibrinous pneumonia, so that even Lænec mentions very numerous examples of the kind, at present its existence is altogether denied. The truth is that it occurs very seldom, yet well authenticated cases have been reported by *Fischl, Leyden* and other very competent and trustworthy authors.

Traube has established the differential diagnosis between abscess of the lung and gangrene. This is to be found in the characteristic *sputa*, in *abscess*, that is, *the constantly present elastic shreds of the parenchyma of the lung in abscess, which are always absent in gangrenous sputa. Leyden* (*Ueber Lungenabscess, Klinisch Wochenbl.*) presents a characteristic description of abscess of the lungs, which he says resembles in many features acute caseous pneumonia, far more than gangrene, although he advises to avoid mistaking abscess for gangrene. He places abscess of the lung under two heads:

1. Perforating, (such as *Stokes* describes under the same name, and *Traube* under the name of latent abscess.) Such are hepatic and peritoneal abscesses situated beneath the diaphragm, or existing in the vertebræ or in the bronchial glands. All of them may perforate the thoracic wall, or penetrate into the lungs, and from there be discharged by expectoration.

2. Pulmonary abscesses proper. Those are abscesses following pneumonia, embolic and metastatic, also local inflammations produced by lodgement of foreign irritant bodies in the lungs. The latter class of abscesses are met in the course of acute pneumonia with crisis on the seventh or ninth day, but which do not lead to convalescence. For a few days after the crisis, the fever will return and expectoration will almost cease for several weeks. But suddenly, after a certain lapse of time, an exceedingly copious discharge of pus will take place, which, as a rule, relieves the patient and causes a return to health. The characteristic sputum from pulmonic abscess is this: It has a sweetish, insipid taste, a slightly pungent odor, and contains shreds of lung-tissue, which are of a yellow or grayish black color, and surrounded by thick pus; fatty acid crystals, hæmatoidin and bilirubin; micrococci different from the very lively moving bacilli found in gangrenous sputa; epithelial lining of the bronchi and air cells, etc.

Its pathological anatomy is yet to be studied. *Leyden* gives some sketchy description of its *post mortem* appearance. "Very frequently," he says, "cavities of various dimensions are found in the lungs, filled with a thick, creamy pus. The cavities have different shapes; they may be single or multiple; they may intercommunicate or not. At the height of the ulcerative process, the walls are uneven, and covered with ragged portions of mortified tissue, of a grayish red, black or black-green. These project into the cavity and are covered with pus. The cavity itself is filled with a creamy pus of a whitish, greenish, reddish or brownish color. In the later stages of ulceration the walls become smooth, and are lined with a pyogenic membrane, of slate color, and covered with pus. The membrane is sometimes very dense, or even cartilaginous, or very thin and fragile. Occasionally partly closed cavities, by cicatricial contraction, are to be met with. Of course such cicatrices cause total obliteration of function of the lung tissue."

"When in later stages of acute pneumonia no absorption takes place, there is formed a chronic interstitial inflammation, which readily passes into *caseous* pneumonia.

"In croupous pneumonia the cellular elements fill only the air cells, the intercellular septa remain unaffected. In the chronic caseous form the intercellular septa become affected. They appear like large bands between the alveoli, for they are filled with emigrated colorless blood corpuscles, and fusiform connective tissue cells, from cell proliferation between the fibres. As long as the inflammation lasts, the vessels of the septa remain perfectly turgid, until the retrogressive process sets in. This begins in the center of the air cells, and spreads externally into the septa, and converts the whole part thus affected (usually a lobule) into a finely granular, half dry, caseous mass."—(*Thierfelder.*) "Under such circumstances the physician will wait for a long while for the resolution of the pneumonic focus, but in vain. For he will find that he has to deal with a case of caseous pneumonia, which will differ in nothing in its consequences, from ordinary broncho-pneumonic phthisis.—(*Rindfleisch.*)

Catarrhal Pneumonia.

Radically different from the fibrinous is the catarrhal form of inflammation of the lungs. As *Virchow* has defined it, the fibrinous partakes more of the hæmorrhagic nature, for the *vessels* only are primarily involved in the disease, whilst the catarrhal is cellular, for the air cells are at once infiltrated with small cells and the alveolar walls are soon affected. The fibrinous gives rise to yellow hepatization, the catarrhal to a variety of hepatizations. The fibrinous follows a typical course, the catarrhal an irregular, atypical. In all forms of catarrhal pneumonia there is bronchial inflammation, only in some the bronchial affection is primary, in others simultaneous or consecutive. There are two distinct forms of the disease, the *Desquamative* and the *strictly cellular*. Both may pass into the caseous, degenerative state.

Until very lately all forms of catarrhal inflammation of the lungs, both acute and chronic, were thrown together under the head of catarrhal pneumonia, or broncho-pneumonia, until *Buhl* established the fact that one form of catarrhal inflammation was really a separate disease, specific in its nature, and having a pathology altogether its own. He designated it in his work "*Lungenentzundung und Tuberculose*," *Desquamative Pneumonia.*

Since then the best clinicists have fully corroborated his statements. The main characteristic of this lesion is the filling up of the air cells with infiltrated endothelial cells in a very early stage

DISEASES OF THE ORGANS OF RESPIRATION.

CASEOUS PNEUMONIA. TUBERCULOSIS OF THE BRONCHI.

TUBERCULOSIS OF THE LUNGS.

Sec. III. *Tab. VI.*

Fig. 3.

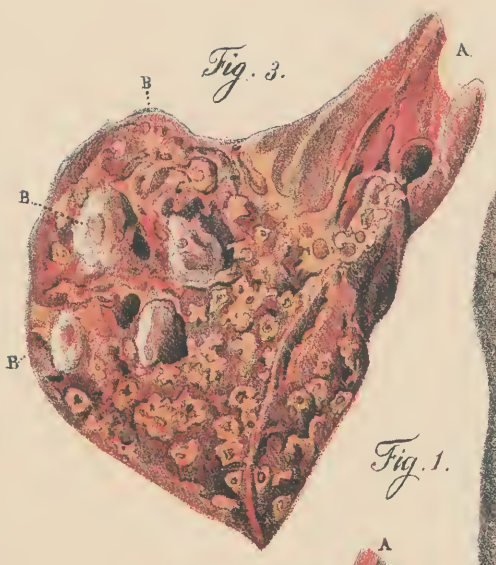

Fig. 1.

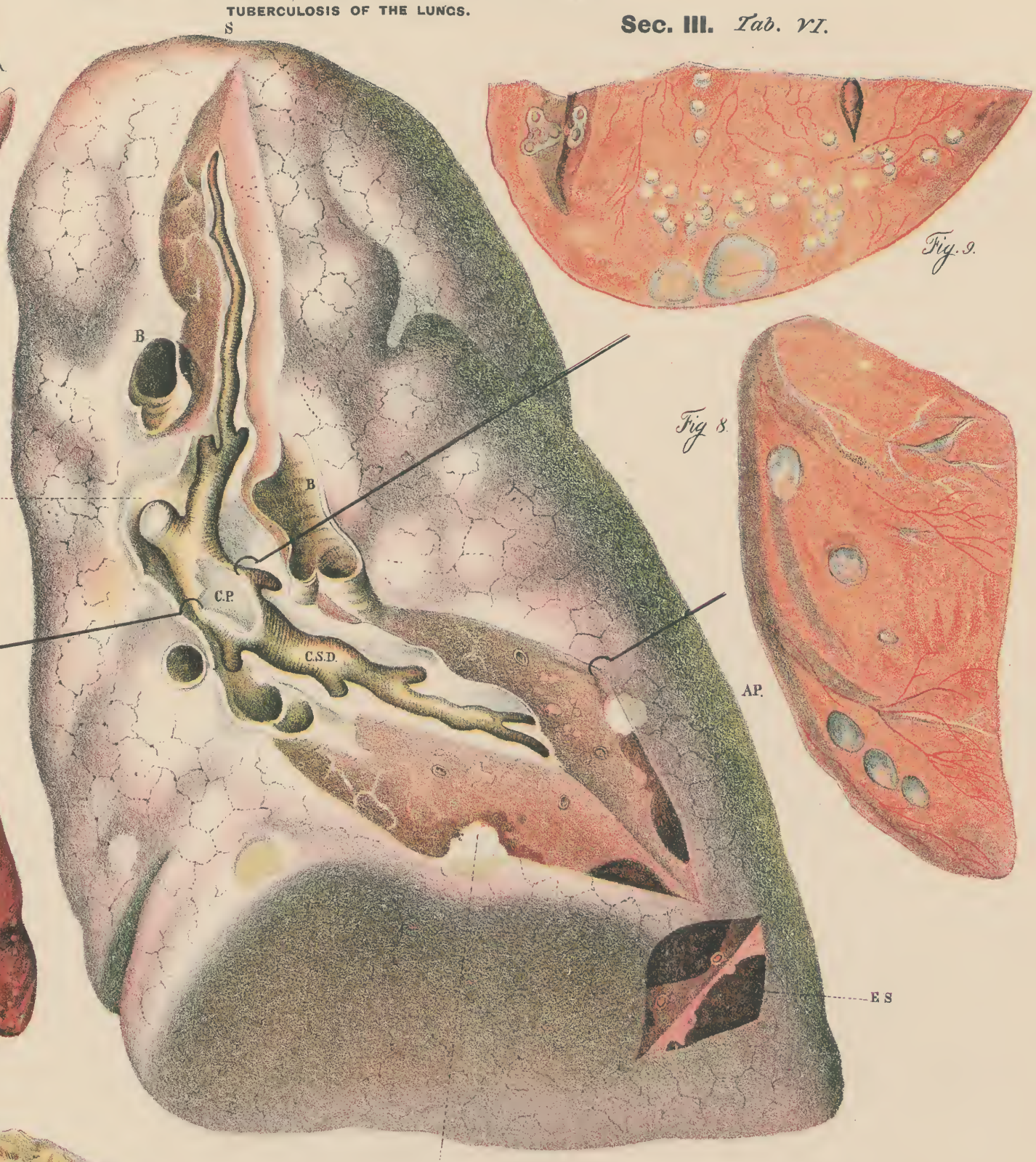

Fig. 9.

Fig 8.

Fig. 2.

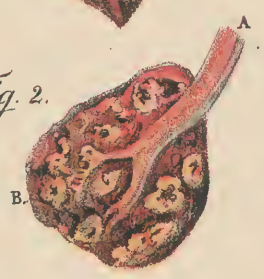

Fig. 7

Fig. 4.

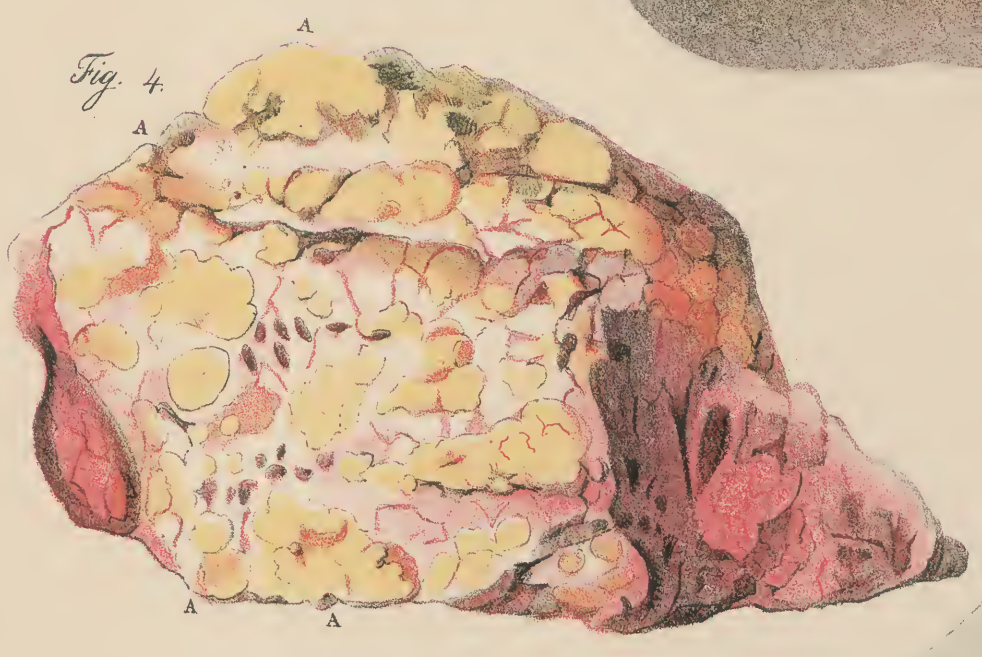

Fig. 6

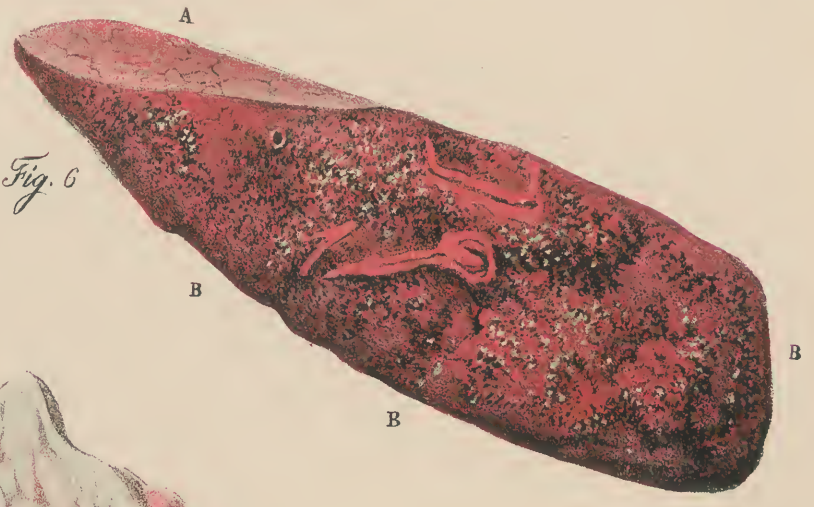

Fig 5

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Sec. III. *Tab. VII.*

Fig. 1.

BRONCHIECTASIS. CAVITIES IN THE LUNGS. ULCERATIONS.

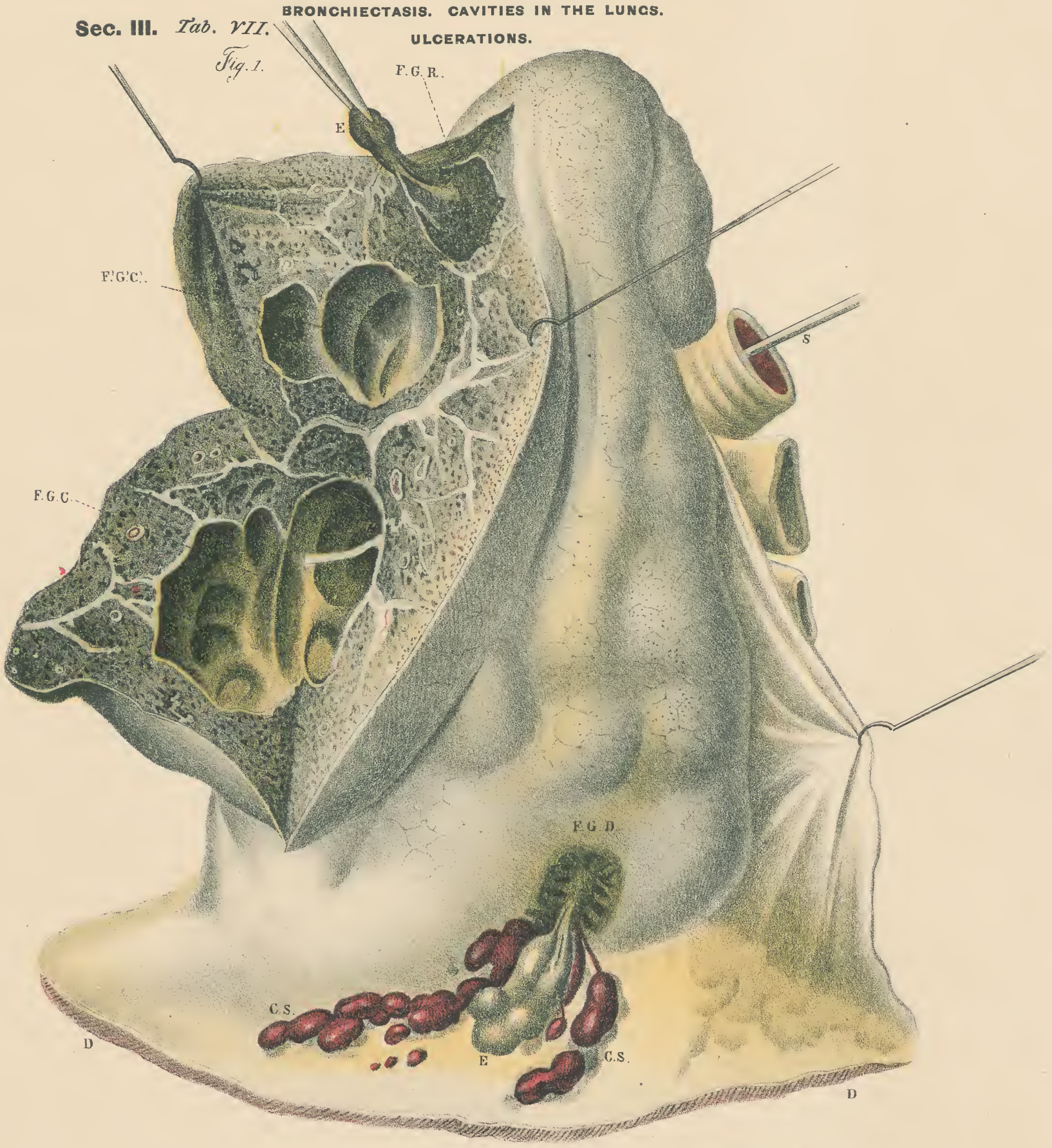

Fig. 2.

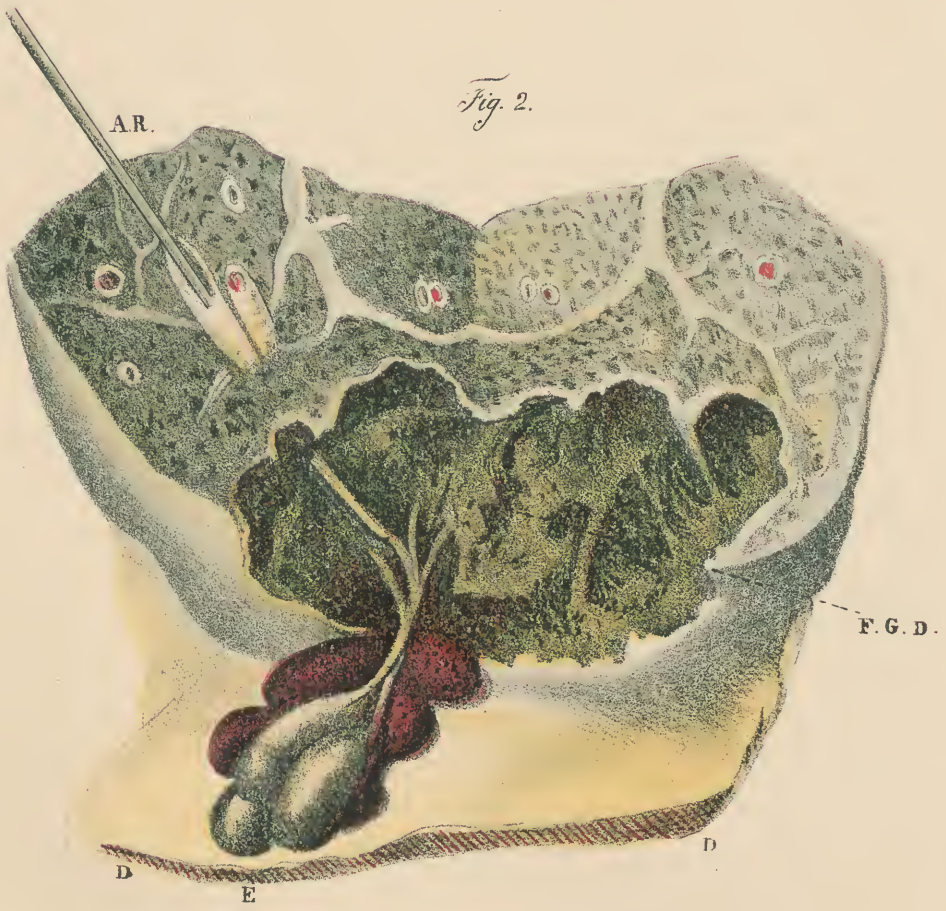

Fig. 3.

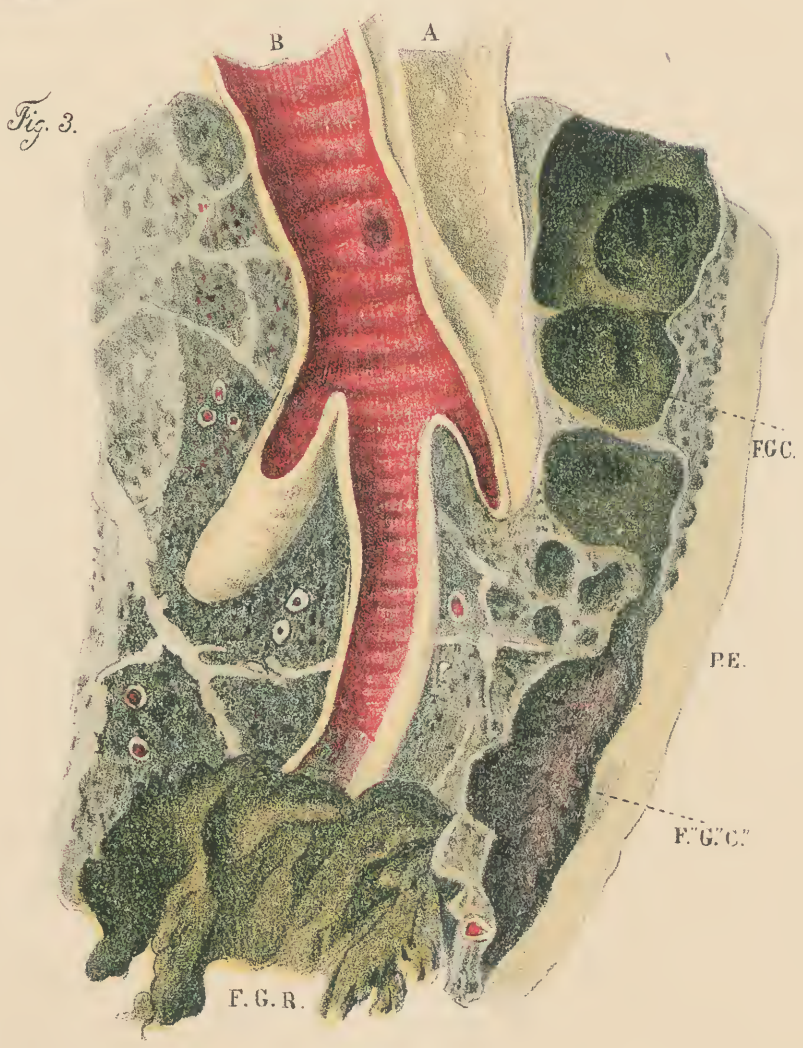

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TABLE VII.

FIGS. 1, 2, 3.—*Bronchiectasis. Cavities formed in the Right Lung. Ulceration and Circumscribed Gangrene.*

CASE.—A man 53 years old.

History.—For years he was occasionally seized with fits of epilepsy. The last ten years he was affected with bronchial catarrh and asthma. He had repeatedly very profuse hæmorrhages and suffered for years with shortness of breath.

Condition and Symptoms shortly before death.—He lies flat on his back, and is almost unable to occupy any other position. His breath is intolerably fetid, and expectorates copiously, large masses of yellowish white, cohesive sputa of an equally horrible stench. Pulse almost normal. *Percussion*: dull sound over the posterior portion of the right lung. *Auscultation*: moist rale on the same side; the left lung sounds normal. *Diagnosed*: *circumscribed pulmonary gangrene.* Excessive hæmorrhage a day later. The blood is foamy, of a bright red, and without odor. The next day, bronchial respiration and bronchophony at the inferior angle and infraspinous space of the right scapula, extending a few inches inward, beyond that region. Dull sound continues; spitting of blood and diarrhea to a greater or less extent for several days. A few days before death he becomes exceedingly weak, is unable to eat, and has a constant desire for strong drink. Dies from exhaustion during an attack of hæmoptysis.

Post Mortem Phenomena.—The whole of the right thoracic cavity is filled with partly clotted and partly liquid blood of a very recent date. The source of the hæmorrhage is a large rent at the base of the right lung, which is attached to the diaphragm (*F.G.D.*, Fig. 1), and from which escaped a quantity of clotted blood and portions of decayed lung tissue into the thoracic cavity. (*C.S.* clots), (*E.*, decayed lung tissue.) By enlarging it, the rent was traced to a gangrenous cavity (*F.G.D.*, Fig. 2), containing sphacelous debris. These proved on examination to be remnants of perivascular structures in a high state of putrefaction. The vessels escaped destruction and served as a support to the putrid mass. A second, though smaller gangrenous cavity, situated in the apex of the same lung, (*F.G.R.*, Fig. 1) of more recent date, contained a ragged mass adherent to the wall of the cavity, and held together by several small arteries. A third cavity (*F.G.R.*, Fig. 2) is visible in the very center of the same lung, a portion of a large bronchus which communicated with it was gradually corroded and partly obliterated. In the thickest portion of the posterior border of the same lung, a large cavity (*F.G.R.*, Fig. 3), very sinuous and multilocular, communicated with several bronchi; the lining membrane of the tube was very dense, fibrous, and of a reddish brown color. It contained no shreds of the tissue, but a quantity of granulating conglomerate extended from the walls, which were shrunk by cicatrization. This cavity was situated beneath the spinous portion of the scapula where the bronchophony was heard. The destruction extended to the pleura (*P.E.*), and had death not arrested it, a communication with the cavity (*F.C.G.*, Fig 3) would have been formed. (*F.G.C.*, Fig. 1) is a cavity in a state of partial cicatrization. Around all these cavities not a trace of acute or chronic inflammation was found. There was no change of color of the impermeable tissue, which was soft, and infiltrated with a brownish serum, which escaped readily on the slightest pressure of the structures. The older gangrenous portions were surrounded by a layer of very indurated tissue (inveterate œdema and dark gray induration). The balance of the lung tissue was soft, of bluish color, and equally much infiltrated Very voluminous fibrous bands of interstitial tissue (Figs. 1, 2, 3) crossed the lung in every direction. The internal surface of the bronchial mucous membrane was intensely red. (*B.*, Fig. 3) shows an opened bronchus. The pulmonary artery and its branches presented a number of plaques on their internal surface (*A. O.*, Fig 3). The vessels in the gangrenous portions were filled with thrombi; in the nongangrenous they were free.

The left lung was nearly normal, so were the brain and the abdominal viscera.

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of inflammation, and differs from the purely cellular infiltration in many of its consequences. It passes invariably into caseous degeneration, especially when inflammation is subacute, and affects a whole lobe.

Catarrhal pneumonia passes certain phases of development, only not so constant as the fibrinous. The phases, according to *Friedlander*, are as follows:

1. Hyperæmia and œdema (*engoument*), which either quickly disappears or passes into,
2. Red hepatization, which might also be re-dissolved and disappear, or pass into either of the following phases.
3. Desquamative, transparent, gray hepatization, which consist of a process of exfoliation of the alveolar epithelium, aud occasional filling up of their spaces. It does not directly pass into the caseous condition.
4. Small-celled or whitish gray hepatization, which begins in the earliest stages of the inflammation, and speedily spreads to the adjacent portions of the lung tissue. This may also pass into resolution when the inflammation is originally not intense; otherwise it invariably leads to *caseation.* The infiltrated portion is at first surrounded by red hepatization, and eventually passes into the desquamative state (SEC. III, TABLE III, Figs. 1, 2).

In the hyperæmic stage the tracheal and bronchial mucous membranes are intensely red, and covered with much mucous and pus. A considerable portion of the lung tissue contains no air, the upper lobe or lobes are usually perfectly infiltrated. The lower lobes are only infiltrated *in spots.* In the air cells there is some serum, some bloated epithelial cells, a few colored and colorless corpuscles and some fibrin. Red hepatization occasionally involves a whole upper lobe; the tissue is not so dense as in the fibrinous form, and is usually intermingled with gray, giving it a marbled appearance. Here and there white dense masses of different sizes project above the red portion, and are perfectly bloodless, opaque and dry. The larger bronchi are usually filled with tough, purulent mucous. A cut surface of a hepatized part will show small yellow spots; they are transverse sections of small bronchiols (SEC. III, TAB. VI, Fig. 7).

Great quantities of fibrinous deposits are found upon the pleural surfaces. The red portions contain red corpuscles, variable quantities of fibrin and small-celled elements and quantities of exfoliated epithelium. In the grayish-white infiltrated portions the air cells are turgid, with densely-crowded small, colorless corpuscles, and compress the interalveolar septa. The capillaries are obstructed. The larger vessels are either partly empty, or filled with a disproportionately large quantity of colorless blood corpuscles. The interstitial tissue accompanying the vessels and bronchi are of double or triple the volume of the normal and filled with the same small-celled elements. The irritation caused by the infiltrate causes the inner coats of the arteries to proliferate, and the space between the endothelium and the elastic laminæ becomes filled with cellular elements. When the disease process is primarily not intense, in the early stages of hepatization, there may still be a prospect for recovery by softening and absorption. But when from the beginning there has been very extensive serous and sanguinous extravasation, the upper lobes especially, are completely infiltrated and contain no air; the lower are in the same condition at the hilus. The affected parts have increased in volume, are tough and heavy. The lower lobes are emphysematous and enormously enlarged. The red hepatized parts are now turned into a gray substance, moist and glistening. Within these are scattered prominent dense masses which have now lost their grayish-white color (SEC. III, TAB. VI, Fig. 6, *B.B.*), and become yellow caseous. They differ in size (Fig. 4, *A.A.*). The bronchi of the infiltrated portion are dilated and filled with a yellow thick pus. The colored blood corpuscles of the red hepatization have now disappeared and are replaced in the air cells by colorless corpuscles and large swollen epithelial cells, easily recognizable by their vesicular nuclei; only a few lymphoid cells are to be found (desquamative stage). As the air cells are not now turgid, the capillaries are filled with blood. The septa are now infiltrated with lymphoid cells, and the air cells contain some serum.

In the grayish yellow infiltrated portion (SEC. III, TAB. IV, Fig. 1.) a condition of far advanced stage of fatty degeneration of the cells exists. The whole structure has lost its contours, and is converted into a uniform caseous mass, involving bronchi, vessels and lung tissue. Close to the pleura such a condition superinduces purulent pleuritis (SEC. III, TAB. IV, Figs. 1, 2).

In the desquamative form we find at this stage an enormous proliferation of the interstitial tissue. It has a distinctly gelatinous, transparent, grayish appearance, especially in the perivascular and peribronchial portions. Under the microscope they present a fine, granulating tissue, and in all stages of development, from the spheroid to the spindle-shaped. Within the septa a number of wide lymphatics, filled with lymphoid bodies, surround the arteries. Cell-proliferation in the coats of the artery commences later than in the perivascular structure, but then it goes on with great vigor. It involves segments of the vessel, and the cellular elements consist of spheroid and large connective tissue cells.

A little later large cells make their appearance in the alveoli, near the exfoliated cells. They are sometimes of a size to fill up a whole air cell, and contain as many as fifty nuclei. They usually occupy the peripherry of the cell (giant cells). The nuclei resemble those of the mononuclear epithelial cells, and probably are of the same origin as these. At this stage the progressive processes of the disease have reached their maximum height. Under favorable circumstances softening of the histological elements and their absorption may take place, and the debris be expectorated. On the other hand, where the bronchi are obstructed by too great quantities of debris, and these are lodged within their cavities for any length of time, dilatation of the cavity ensues. In the caseous portions no change takes place. The tissue becomes more and more atrophic from lack of nutrition; the affected portions approach each other by shrinking of the atrophic tissue, and the bronchi become more and more dilated and filled with turbid liquid pus.

Although desquamative pneumonia invariably passes into caseous, it does not necessarily follow that the latter is always produced by the former. The transition of the desquamative into caseous is brought about when from any cause (such as obstruction of the vessels, or in the bronchi) the exfoliated epithelial cells cannot be removed from the center of the air cells; they undergo necrotic change, and though they may liquify, the same obstructions will prevent their elimination or absorption. This will cause a further exfoliation from the walls of the air cell, and a still further degeneration of the newly-exfoliated epithelium, which will accumulate as an

TABLE VIII.

Embolic Pneumonia. Infarct into the Lung Tissue. Stenosis of Left Auriculo-Ventricular Opening, with Hypertrophy of the Left Ventricle.

CASE.—A woman 44 years of age.

History.—Had frequently very violent palpitation of the heart, lasting several days; experienced constantly in the region of the heart a sense of oppression and suffocation; was repeatedly attacked with hæmoptysis.

Condition and symptoms three days before death.—Face purple; expectorates clear blood, which is sometimes bright red, sometimes dark colored; very hurried breathing, but claims not to feel oppressed; is able to speak audibly; pulse almost gone; beyond some moist rale in the bronchi, nothing very abnormal in the lungs; beating of the heart very tumultuous, yet without murmur.

Is gradually overcome by weakness; coma; involuntary discharges; dies from suffocation.

Post Mortem.—Hæmorrhagic infarcts disseminated throughout both lungs, some very extensive, some small (Figs. 2, 3). They were irregularly spheroid and distinctly circumscribed, their black color contrasted with the lighter colored surroundings. The greatest number of the infarcts were immediately below the pleura, which they raised in several places; they presented a friable, granular appearance, and seemed to have obliterated all the air cells, some of which were torn by the massive effusion of the blood. The lower lobe of the right lung looked like one affected with fibrinous pneumonia; it was yellowish, red and granular, but offered that difference, that it was highly compact and did not crepitate. There was hypertrophy of the left ventricle and stricture of the auriculo-ventricular opening of the same side to a marked degree.

FIG. 1, Melanotic pneumonia and anthracosis over a large portion of the outer surface, and within the lung, of a man who worked in a coal mine. The particles of coal-dust had mingled with a quantity of pulmonic pigment, and formed circular spots immediately below the pleura. There were a number of fibrinous deposits upon the membrane, in the shape of strings and circular spots, some of which looked gray, others white. The interlobular tissue was hypertrophied and plainly marked the interlobular boundaries.

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irritant detritus in its cavity; and when the watery portion of this dead mass is absorbed by imbibation, a central caseous debris will remain.

Caseous degeneration in small-celled hepatization is produced by the anæmia caused by the over-filled air cells with these elements. For, no circulation can take place when the vessels are compressed.

The small cells are of a triple origin:

1. From the endothelium of the vessels.
2. From the blood in the artery.
3. From the internal coat and the vasa vasorum.—(*Friedlander*).

Strange as it may appear, that the cells should be derived from the cavity of a vessel, whilst the blood is in a state of circulation, yet *Bubnoff* has clearly demonstrated that migratory cells pass from the cavity of arteries, through their walls, outside; and *Saviotte* has also seen those migratory cells enter capillaries whilst blood was in full circulation within them. Where vessels are thus affected, they become unfit for nutrition of tissues.

When caseation has once taken place, it may remain in the tissue a long while without undergoing any change. (This will often prevent differential diagnosis between acute and chronic catarrhal inflammation.) *Rindfleisch* considers the caseous state as a pathognomic sign of the chronic form. The older pathologists have taken this caseous condition as a form of pulmonary phthisis.

Although this is not tuberculosis, it is a state from which tuberculosis may readily develop; but it does not always do so. Caseous masses differ, in the lungs, in color and consistence, according to the mode of their production. The dry form is yellowish white, tough, slightly transparent and constitutes a form of coagulating mortification. This is usually found in the proximity of the smaller bronchi. A thorough caseous mass is always without a nucleus, sometimes perfectly homogeneous, sometimes slightly granular. The homogeneous often passes into the granular form. The soft variety is white, and consists of a fatty albuminous detritus. Both forms may exist by the side of one another, or the solid variety may pass into the soft. The final termination of a caseous focus is either *colliquation* and eventual absorption, or transformation into a calcareous mass. The lime in this is generally united with albumen; when decomposition takes place the albumen is eliminated, and a very brittle chalky mass is left.

Acute catarrhal pneumonia clinically differs from its chronic form only in extent and not in kind, for both have the same anatomical basis; and this is inflammatory or irritative alteration of the mucous membrane of the bronchiols. They both have hyperæmia and œdema of the tissue for a beginning, and both may be considered primarily as *bronchiolitis*. It only depends on concomitant circumstances to assume the one or the other form. In both there is alteration in the cavity and wall of the bronchi and bronchiols, also in the pulmonic tissue proper.

When the muco-purulent secretion stagnates in the bronchial tubes, it becomes gradually inspissated, its cellular elements undergo molecular death, and the whole constitutes a yellowish white softish nodule, indicating the former cavity of the tubes which are now converted into solid cylinders. Such a condition can but cause excessive irritation to the bronchial wall, and, sooner or later, reactive phenomena of its tissue will become manifest in the mucous membrane at first, in its other structures subsequently. The peribronchial tissue will undergo a kind of callous indurative change, after the progressive hyperplastic processes of inflammation have involved the tissue proper of the tubes. Such thickened and filled small bronchial tubules will present the appearance of solid nodules, which were formerly held to be tubercles.

The thickening of the peribronchial tissue is associated with a similar condition of the interlobular connective tissue, its immediate continuation. The accumulated secretion may become so abundant as to dilate parts of the smaller bronchial branchlets and to attenuate their walls by pressure and atrophy. When a number of branches of the bronchi are thus partly closed up and dilated, the inspired air will have a tendency to dilate the still open portions of the respiratory organs, and permanent dilatation may thereby be produced. This ectatic condition must not be confounded with the highly hyperplastic and widened bronchi found in common catarrhal bronchiectasis. In the bronchopneumonic ectasis only the mucous membrane retains more or less its normal character; the other tissues of the tube are exceedingly atrophied. More especially do the blood vessels partake of this atrophic condition. The catarrhal secretion abounds in cellular elements and contains but little liquid, and is firmly attached to the wall of the tube in little heaps. Eventually the mucous membrane may undergo the process of ulceration of a highly destructive nature, and cause extensive excoriation of its layers. Frequently tuberculous matter is found in such ulcerated tissue. When such a state of the bronchial tissue continues for any length of time, or when the ulcerative process is repeatedly reproduced, destructive action extends to the pulmonic parenchyma, which is generally by this time already unable to perform its function of renewal of air, and formation of more or less extensive cavities begins. The effect of the closed bronchi upon the lung tissue—besides the histological changes it superinduces, as described before—is the formation of a condition similar to fœtal atelectasis. The infundibula become contracted, the air cells shrink, and the parts situated on the outer surface become uneven. The shrinking of the air cells necessarily brings the blood vessels closer to each other, and this gradually superinduces a permanent hyperæmia of the parts. Sooner or later this hyperæmia produces exudative œdema in the now shrunk tissue. This serous infiltration once more swells the tissue up, but makes it soft and very compressible. Outside it has a bluish color; on a cut surface the color is deep reddish brown, moist and smooth, and resembles very much the pulp of the spleen. It has received the name of splenisation. Such splenisation always indicates a low heart power associated with static hyperæmia and serous infiltrates in the airless alveoli.

A state like the one just described may terminate in two ways: Either *inveterate œdema* or *gray induration*. The first differs from a splenoid state, that no hyperæmia can take place in the now exceedingly infiltrated tissue, which seems impermeable to the solider portions of the blood. This seeks new channels in more yielding portions of the structure. The œdomatous tissue looks pale or slightly rose color.—(Sec. III, Tab. X, Fig. 2, *L. S.*)

From a cut surface of such a lung, a clear, highly concentrated serum, containing yellowish white particles, oozes out. Under the microscope the particles prove to be fatty degenerated cells, collected in minute nodules. The surrounding hyperæmic tissue strongly contrasts with the pale structure.

Gray induration is regularly found in the above described chronic peribronchitis, which is ever connected with an inflammatory hyperplasia of the interlobular septa. These become the most prominent part of the otherwise atrophied parenchyma.

The air cells are nearly empty, or altogether wiped out by the neoplastic interstitial tissue. Only the interlobular vessels are still to some extent permeable. The gray or blackish blue color is due to collections of black pulmonic pigment, which is found in variable quantities in its different portions. (Sec. III, Tab. VIII, Fig. 1, Sec. III, Tab. IX, Fig. 1, *M.P.P.*, Figs. 2, 3, *L. S.*) Originally the pigment constituted the remnant of exceedingly numerous minute hæmorrhagic collections. Mingled with these are great numbers of particles of coal dust or soot inhaled and deposited in the tissue (Sec. III, Tab. X, Figs. 1, 2, 5).

Ultimate Changes of Caseous Masses.

The greater the volume and the quicker the formation of caseous masses, the sooner do they undergo retrogressive change, which always begins in the center of a mass. By attraction of water from its periphery, it softens, turns into a pus-like, flocculent, semi-solid substance. When corrosive inflammation has preceded the softening, which happens most frequently in dilated bronchi, clogged up with stagnant masses of secretion, etc., the parenchymatous as well as the bronchial tissue will become disintegrated and cavities will form. The cavities will generally contain exceedingly corrosive substances, which, when they pass through any orifice formed into a bronchial tube, form communications with the outer air. Putrefactive agencies soon penetrate such cavities, and convert the greatest portion of their contents into exceedingly putrid material. These are generally expectorated as highly offensive sputa, containing remnants of pulmonic and bronchial tissues, in all stages of decay. As long as such sputa are thrown out, that long we may be sure that a cavity or cavities are extending, for they are the debris of destruction of their walls. Reparative process in an infiltrated caseous tissue can only take place after *the dead caseous* mass has been eliminated or changed, chemically, into an inert substance.

HAEMORRHAGIC PNEUMONIA.
SPLENOID LUNG-TISSUE.
BROWN INDURATION.

Sec. III. *Tab. VIII.*

Fig. 1.

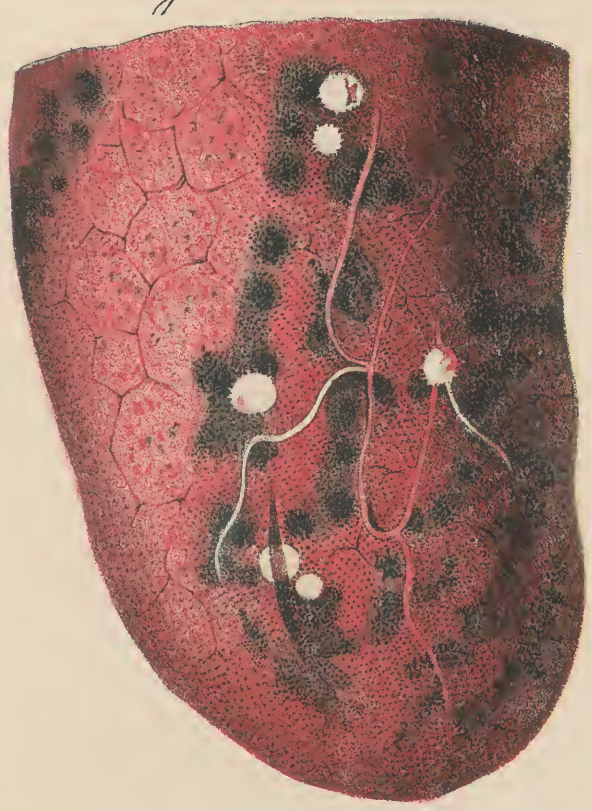
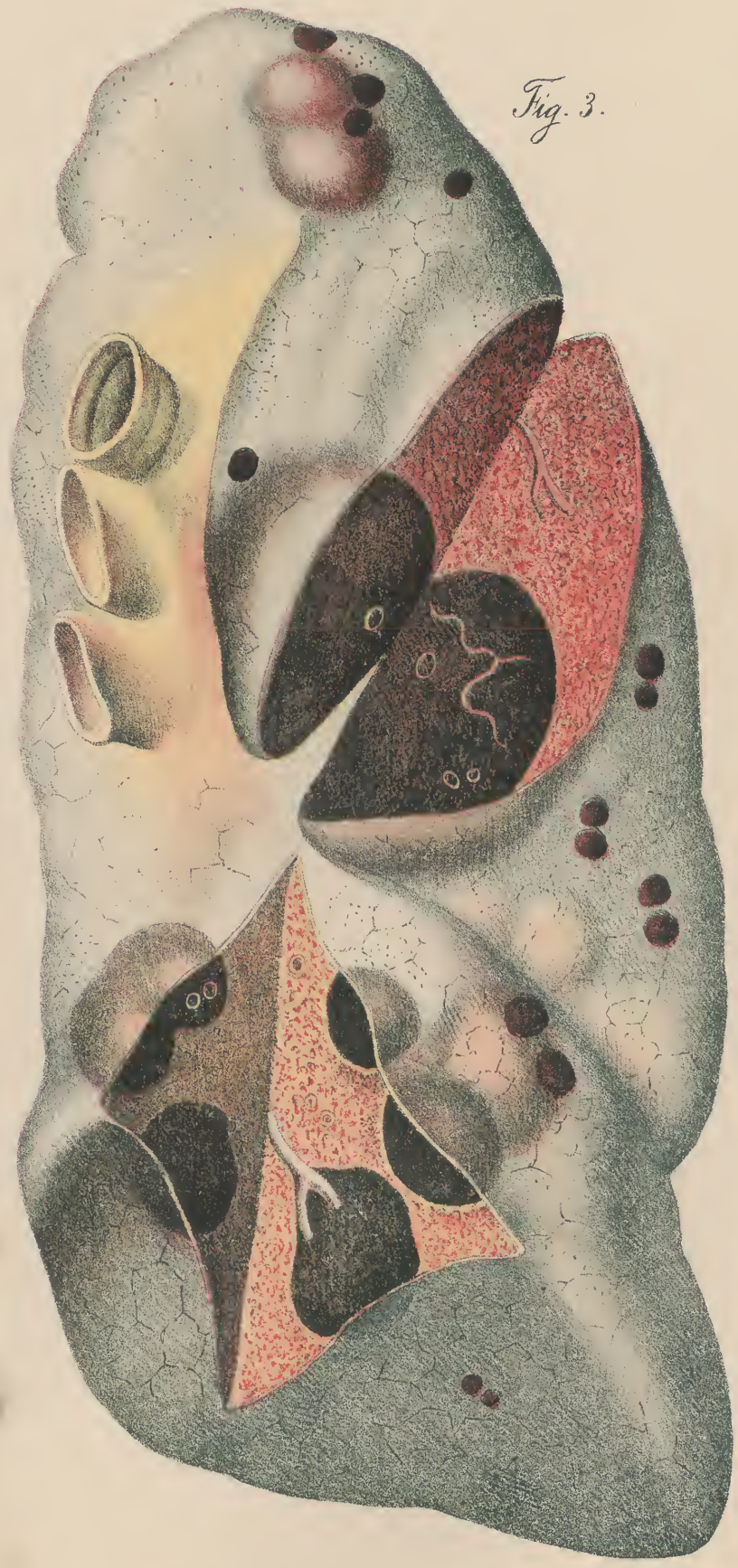


Fig. 2.

Fig. 3.



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DISEASES OF THE ORGANS OF RESPIRATION.

CAVITY IN UPPER LOBE OF RIGHT LUNG.

ADHESIVE PLEURITIS.

Sec. III. *Tab. IX.*

Fig. 1.

Fig. 4.

Fig. 2

Fig. 3.

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TABLE IX.

FIG. 1.—*Cavity in the upper lobe of the right lung. Adhesive pleuritis. Infiltration and tubercles in the left lung.*

CASE R.—A man 30 years of age.

History.—Had a dry, harassing cough a number of years. Lately he presented the following symptoms: A cavity in the apex of the right lung. *Percussion:* tympanitic sound over the region of the cavity. A singular vibrating murmur at the end of every expiration heard on auscultation. Latent extensive pleuritis on the same side; thorax distended over a large portion of the right lung, indicating exudation by dull sound over infiltrated part. No fever. Has an enormous appetite. Shortly before death he could not be up, but had to lie on his right side. Was slightly anasarcous. Was suddenly seized with very severe pain in the right axilla, where enormous phlegmonous inflammation had set in. Tumefaction and pain extended forward beneath the pectoral muscles and backward beneath the serratus magnus; also all along the whole arm. Died six days after this attack.

Post Mortem.—The whole axillary cavity filled with pus, and extending forward and backward. The vascular and neural sheaths, the perivascular tissue, and the muscles were all infiltrated with pus. Large quantities of it were contained in the folds of the fascia.

The right lung (Fig. 1) was reduced in volume and collapsed. Was surrounded by a pseudo-membrane and extensive exudation. The upper lobe of this lung was firmly attached to the costal pleura; the balance of the lung was separated from it by massive exudates; the base was enormously excavated. No part of the organ was permeable to air. The tissue was soft, infiltrated with a turbid liquid, and penetrated in every direction by gray colored fibrinous bands. A few indurated tubercles were scattered through the tissues. The superior portion contained a cavity, which was partly filled up by two conical fragments of lung-tissue (M. P. P.) They were indurated, but the substance to which they were attached readily broke down. The walls of the cavity were very thin and communicated with some bronchi, which were much corroded, and in the cavity of which extensive ulceration existed. The apex of the left lung showed a fine example of cicatrization of caseous tissue. This was shrunk, wrinkled, and indurated. Between the dark-colored structure white bands extended in all directions, giving it a striped appearance. A great many indurated tubercles, of various sizes, were disseminated throughout the whole mass. (F. M., mediastinal surface of the pleura.)

Fig. 2 represents a vast cicatrized cavity (C. P. C. P.), occupying the superior lobe of the right lung of another individual. Great numbers of bands and strings traversed it in all directions. They consisted of cicatricial tissue, and on their surfaces presented the appearance of mucous membrane. Each band contained very numerous blood vessels. In the bronchial cavities, which communicated with the excavated lung, there was no mucus. A few smaller cavities (C. P. C. P.) partly closed by cicatrization existed in the lower portions of the lung.

Fig. 3 shows an excavated upper portion of a lung by ulceration, and sinus opening externally at the left lateral portion of the larynx. (F. S., pulmonary sinus.) (L. S., superior lobe.)

Fig. 4. A highly thickened portion of the pleura, in the case described in Fig. 1. (A. A., external surface.) (B. B., mediastinal surfaces.) (C., apex where the pleural surfaces meet.) The membrane is covered with fibrinous ridges and other neoplastic formations.

The work of repair begins in the pleural and in the interlobular tissues in the following manner: Either a pyogenic membrane is formed by the interlobular connective tissue round a caseous lobule, and thus cuts the communication between the affected and the non-affected parts, the afferent bronchus and the blood vessels forming a sort of a scaffolding to support the cyst-like enlosure (*Rindfleisch*), or, a granulating membrane is formed by the interlobular structures surrounding the caseous mass. Vascular spurs, enveloped in great quantities of granulating tissue, filled with pus corpuscles, enter the cavity, and fill it gradually up from all sides with a stiff cicatricial tissue. The great vascularity of such tissue in its formative stage often gives rise to frequent, though small, hæmorrhages. When the cavities are not large, or do not communicate with other cavities; or, when the caseous formations are confined to few localities, the granulations produce contracted cicatrices, the lung tissue shrinks and becomes harmless. Occasionally a whole series of partly obliterated cavities are found in a large portion of a lobe, the central portion taken up by the obsolete vessels and bronchi (Sec. III, Tab. IX, Figs. 1, 2). When a reactive inflammation sets in, in a very early stage of the caseous process, by extensive hyperplastic action, very voluminous and very dense bands of connective tissue of various colors are formed. They begin at the thickened pleural covering, and traverse the organ in every direction, and connect with the perivascular and peribronchial tissue, thus forming a labyrinthine system of cavities and passages. Each is invested with a pyogenic membrane, and is separated from the adjacent cavity by a very thick membranous partition. The bronchi in such structures are in a high state of dilatation.

The usual result of the softening of caseous matter, and concomitant ulceration, is the formation of enormous numbers of abscesses, which follow each other in succession until the whole mass, when it becomes colliquated, is emptied into the pleural cavity, and produces pneumo-pyo-thorax (Sec. III, Tab. VII, Figs. 1, 2, 3). Such dangerous and usually fatal termination is sometimes prevented by thickening of the walls of the abscesses and attachment to the pleural covering.

Tuberculosis.

Considering the great frequency of tubercles in the tissues of man and animals, and the innumerable researches, both clinical and anatomical, made by very numerous of the most able pathologists and pathological anatomists into this subject of Tuberculosis, one might expect that, by this time, a well-defined notion of its nature and morbid character, or at least a definite pathological anatomy of the lesion has been established. Yet such is not the case. It seems even as if the multiplicity of the labors of the investigators has rather tended to confuse than to solve the problem. The great reason of the vast diversity of opinions must undoubtedly lie in the extremely variable phenomena manifested under different conditions by the morbid processes peculiar to tuberculosis. In a work like this, having a compendious character, only what has been definitely established can find place on its pages, and only so much as is so far certain will be described.

The main character of Tuberculosis constitutes the presence of a greater or lesser number of various sized cellular nodules in any involved tissue. These nodules have been, and still are, called tubercles. Formerly all sorts of infiltrates and other morbid formation, having a knotty appearance, were designated as tubercles. Baillié (in 1794) and Bayle (in 1810) first called attention to the gray nodules or congeries of cells, which we at present call tubercles. Bayle extended the name tubercles to many other forms of pulmonary phthisis, and introduced thereby great confusion into the true nature of the disease. Afterward Laenec designated the caseous masses found in many diseased lungs as TUBERCLES; larger infiltrates occupying a great portion of, or a whole lobe, he named *Tuberculous Infiltrates*, and the true tubercle he called *Miliary Granulation*. With him, and for a long time afterward, a caseous condition of a tissue was considered tuberculous. Virchow has subsequently shown that caseous masses may form in various ways and have different significances. On anatomical grounds he established the *Cellular Tubercle*. At present a tubercle is defined as an avascular cellular nodule, which only reaches a certain size, and when arrived at the height of its development remains for some time in that condition, and then becomes caseous, which then undergoes either softening, mortification, or becomes calcareous. The most recent investigations of *Langhans*, *Schuppel*, *Koester*, *Rindfleisch*, *Conheim*, and *Ziegler* have added the facts that the tubercle has in many cases a peculiar histological structure; that the same kind of cells often recur in the tubercle and give it a specific character. The central portions of it contain giant cells, which are many-nuclear. Single nuclear cells resemble lymphoid elements. Sometimes they are of large size, and have an epithelioid form. The larger cells have a largely granular protoplasm and small spheroid nuclei. They are usually imbedded in a *stroma* having a net-like arrangement. At one time it was thought that the epithelioid and giant cells were peculiar to tubercular structure, and they were considered diagnostic of that morbid alteration; but it was found that every inflammatory tissue-formation is preceded by a stage of development of large cells, and a number of tubercular elements can readily be produced experimentally. *The cellular elements met with in the tubercle are likewise found in every granulation;* and everything speaks in favor of the idea that a *tubercle is a specific form of granulation, but none of its elements are special to it*, for they, like all granulating elements, are derivatives of the emigrated colorless corpuscles from the blood vessels; the fixed elements of the tissues contribute but few contingents to the make-up of the tubercle. Histologically they differ from ordinary granulations only in that they contain a greater number of large or epithelioid cells, assume a nodular form, attain a certain stage, and undergo retrogressive change.

Tuberculous nodules are met in diseased tissues in all the stages of progressive and retrogressive development, and may contain either larger and smaller cells grouped together, large cells alone, or small cells only in one tuberculous group.

A tubercle, according to the latest investigations made by Robert Koch, of Berlin, will have to be defined *as a cellular nodule, in which there is contained the specific virus, consisting of the Bacillus Tuberculosis*. Whether this will become the permanent definition or not the future will decide.

Propagation of Tubercles.

Tuberculosis coming within the category of infectious granulations, it partakes of the nature of these specific inflammations as Rindfleisch has properly characterized them. Clinically considered, they have the peculiarity of being infectious in a variety of ways: By progressive extension, that is, by passing from a central point centrifugally into the adjacent tissues. Simultaneous with this extension the central infectious portion undergoes a process of decay. The lymphatic system connected with it becomes involved, and within the lymph-glands similar infectious centers are formed, from which further extension into the different parts of the

TABLE X.

Bronchitis and Peribronchitis.

FIGS. 1, 2, 3, 4.—PUTRID BRONCHITIS.

The bronchial branch (A) has been partly obliterated by ulceration. Its color is purplish, and shows that only a portion of its mucous membrane remains unaffected. The lung-tissue is dark gray and indurated, showing some very dark spots and stripes mingled with it. Figs. 2 and 3 represent the pleural covering of that lung; shining, black little tumors, being melanotic, infiltrated lobules projecting upon the surface of the lung immediately beneath the membrane. Fig. 3 shows an emphysematous state of the lung; Fig. 4 a highly dilated bronchus with portions of emphysematous lung-tissue.

Fig. 5. A horizontal section of a large bronchus. A portion of peribronchial tissue (A) is nearly normal, while (B) shows enormous dilatation of the lung cells, and infiltration with tubercular matter in the infundibular septa. The bronchial glands (C) are exceedingly enlarged, filled with dark pigment, and in a partly ulcerated state.

Fig. 6 shows a small portion of a bronchus filled with a great number of crystals of cholesterine and some phosphatic masses, colored red by haemoglobin and other blood-pigment (magnified).

Figs. 7, 8. Portions of lung-tissue near the involved bronchi in which all traces of normal lung-structure are obliterated. In both they present nearly homogeneous textures. Fig. 8 a closed large bronchus, forming a shallow cavity filled with partly organized connective tissue. Some traces of a former caseous state are still left here and there, indicated by slight depressions in the level of the tissue.

Figs. 9, 10. The apex of a lung in a state of vesicular emphysema. The bronchi are partly filled up and partly ulcerated; the peribronchial tissue is nearly destroyed, and in its place cavities filled with dark purulent masses are to be found. Fig. 10 is a magnified view of the bronchial and peribronchial alterations (A. A. A.) emphysematous tissues, showing enlarged lobules surrounded by highly infiltrated septa, whilst very much dilated blood-vessels form a net-work upon the face of the visceral pleura. The bronchi (B) are nearly denuded of their epithelial coverings.

Figs. 11, 12. Two very large calcareous masses, found in the right and left upper lobes of tuberculosed lungs. Perfectly altered bronchus (A), indicated by a somewhat lighter color than the rest of the mass. Thickened pleura (C). Fig. 12 has a distinctly fibrous texture, and is of more recent origin than Fig. 11. It shows a depression made in its upper portion by the first rib. The individual in whose lungs these masses were found was old and decrepid; had led a very dissolute life.

organism are brought about. As in the greatest number of infectious granulations, so also in tuberculosis is the morbid process propagated upon other or foreign organisms, as innumerable examples have proven its hereditary as well as its infectious transference upon other individuals besides the one primarily infected with it. Very seldom is the primary stage of tuberculosis in any organ ever met in anatomical examination. Usually the advanced stages are found, and the parenchymatous structures have already undergone great modifications. Only in experimental pathology are the initial states to be observed, and that only in animals. Once in a great while, by the side of a compactly infiltrated portion of tissue, a caseous nodule in its earliest phases may be observed. It will then be found to consist of the elements above described, and will have a fairly transparent look, and will resemble a portion of ordinary granulation on the surface of the skin when epithelial cells are developing in large quantities. In its later stages, as usually met with, the parenchymatous organs contain caseous nodular foci, which are either thoroughly compact or already softened in their centers. In superficially situated tissues ulcers are formed in the softened excavations. The edges and fundus of those cavities are caseous; surrounding these is a gray or grayish-red slightly transparent zone of structures, resembling very much ordinary granulating tissue. This same class of tissue is to be found in different portions of the affected organ in greater or lesser patches; they usually contain small gray or yellowish-white opaque nodules. Likewise are there small grayish little knots in that class of tissue. They are surrounded sometimes by hyperæmic portions; some by apparently unaltered structures. The first are usually plainly visible to the naked eye. The grayish-red or the gray transparent masses, which surround the caseous portions, or form the fundus of an ulcer, or constitute solitary centers are nothing else but granular infiltrated tissue. The yellow nodules enclosed within those are either fresh giant cells containing tubercles, or older ones already in a caseous state. The differential diagnosis between granulating tissue and tubercular masses is readily effected by tinction of the tissue with two or more colors. The epithelioid and giant cells are not nearly as readily stained as the small spheroid ones. In a tubercle three different zones are produced by the coloring process. The innermost portion is formed of nuclear parts of the giant cells, which assume a darker color than the zone immediately surrounding them, which consists of the epithelioid, and is much lighter. The most external are the spheroid cells; they are more intensely colored than the rest of the granulating cells. When in the center of a tubercle caseous alteration has taken place, giant cells are superposed here and there. Sooner or later the lymph-glands nearest to a tubercular center become involved, and a tubercular eruption near the old center is formed. From the nearest lymph-glands a progressive spread of tubercular formation is extended into larger lymphatics and eventually are carried into the thoracic duct, and thence into the blood circulation. Within the lymph-gland tubercular eruption is most abundantly developed, and whenever any favorable conditions will be present tubercles will form. With the eruption of tubercles there is always associated a more or less intense inflammation of its surroundings, recognizable either by hyperæmia, or by infiltration into or swelling of the tissue surrounding the tubercle. When this process goes on for a while, there is formed new connective tissue in the place of the tubercular eruption. The termination of the secondary tubercles is caseation and decay; very seldom do they turn into connective tissue, more seldom does absorption of tubercles take place. When tubercle-producing agencies, from a decaying tuberculous mass existing in a lymphatic gland, or in a tubercular ulcer in the thoracic duct, enter into the blood-current they disseminate in the several organs of the body. The same dissemination is brought about by tuberculous infectious matter entering directly into the blood vessels. Such blood infection will be followed by eruptions of tubercles in single organs of the body, or in all the internal organs, or, at least, in most of them, constituting a condition medically called "*Miliary Tuberculosis.*" The nodules are found in the organs, of a size ranging from that of a mustard seed to four or five times its volume. The smaller ones are grayish, jelly-like, and translucent; the larger are opaque and yellow. The latter are usually caseous in the center. When single organs are affected, they offer very nearly the same morbid appearance. In the early stages of their development they are made up of small spheroid cells, and directly derived from the blood, as can be proven experimentally. Some tubercles retain their cellular character to the last; they only increase in volume as they develop farther. Some contain giant cells and epithelioid. All terminate in caseous degeneracy. They are very seldom converted into fibroid tissue or are absorbed.

Connected with eruption of tubercles are always very diffuse and extensive inflammatory derangements of the circulation. In the lungs they are also associated with extensive exudations.

Miliary tubercles are invariably developed from emigrated blood corpuscles. Occasionally the fixed cells assist in their formation. When tuberculous *virus* enters directly into a blood vessel, it causes destruction of the vascular walls by formation of tubercles in its several coats. Sections of tubercular lung tissue show very numerous tubercles in the walls of its vessels. That all tissues of the body are not alike affected by tuberculosis, or like the skin not at all, is no doubt due to inequality of distribution of the blood in them, and partly to peculiarities of the tissues themselves.

Miliary tuberculosis is not necessarily the result of organic tuberculosis. As a rule, the tuberculous process does not pass the limits of the primarily affected organs and their accessory lymphatics. Decay of caseous tubercle of lymph-glands leads soonest to infection of the blood. Mucous surfaces are, at times, also the propagators of tubercles, and extend them to surfaces of and into other mucous tissues, with which they are in anatomical or even physiological connection. Thus do, for instance, tubercular lung tissues infect the tracheal, laryngeal, pharyngeal, and sometimes even the upper intestinal mucous membranes with tubercles. The serous membranes are even better carriers of tubercular infection than mucous membranes. The same as the parenchymatous organs are some mucous membranes more liable to be infected than others. That of the mouth, the pharynx, and the æsophagus are far less liable to become involved than that of the larynx and trachea. The stomach, the duodenum, and the biliary ducts, also the urethra, are hardly ever affected with tuberculosis.

Pathology of Tuberculosis.

The above described morbid processes, which are all comprised in what is named Tuberculosis, are anatomically characterized by formation of nodules, and clinically by their successive invasion of one or several organs, or of the whole organism; farther by producing progressive destruction, not only of the parts which are primarily affected, but also of those which become involved by infection from the former, in the different modes of propagation. It is also characterized by a more or less continuous process of inflammation in the different organs involved, the peculiarity of this form of inflammation consisting in the production of anatomically well-characterized morbid structures, the tubercles *cellular and avascular nodules.* Besides the above pathological features there is also a distinct mark belonging to it that it is transferable to other individuals, both animals and men, as Villemin and Klebs have at first clearly shown by experiment, and has been confirmed by very numerous other investigators (Waldenburg, Conheim, Bollinger, etc). The tubercular agency is both inocculable and transferable, in its fresh state as well as in the caseous condition, and produces the same characteristic anatomical, and to a great extent clinical peculiarities as the ones existing in the individual from which the infection is derived. It is, in fact, an infectious disease. That not all animals are alike infected by inoculation is true; also that not in every kind of animal are tubercules reproduced is also a fact. This only confirms its infectious nature, that it attacks certain kinds and classes of animals, and of these kinds only certain individuals. In man clinical experience has proven that members of one family are not all alike affected by what is called an inherited tubercular *taint.* Also that some persons cohabiting with tuberculous individulas

BRONCHO,PNEUMONIA.
BRONCHITIS. PERIBRONCHITIS.

Fig. 1.

Fig. 2

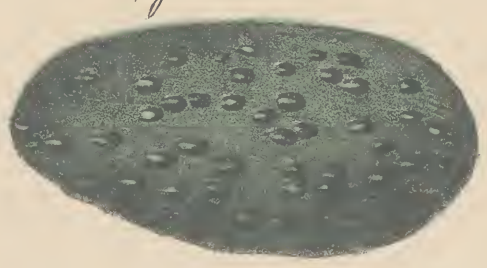


Fig. 3

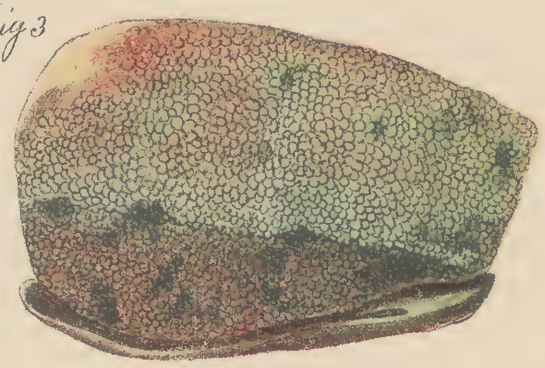
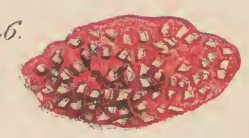


Fig. 4.

Fig. 6.



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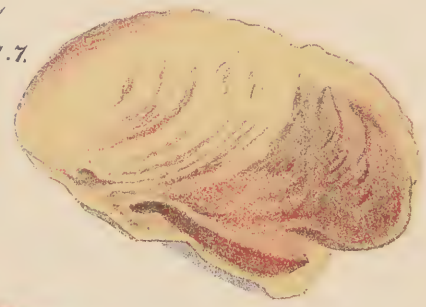

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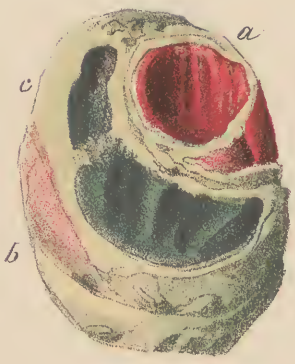

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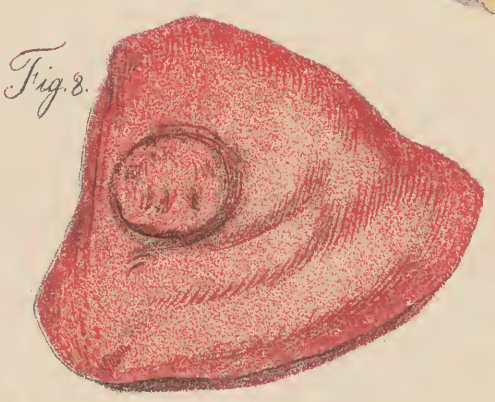


Fig. 9.

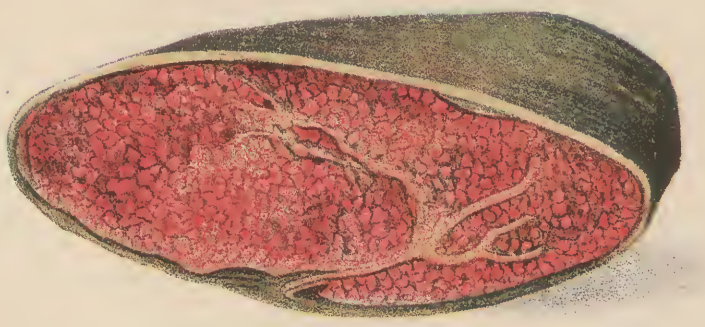
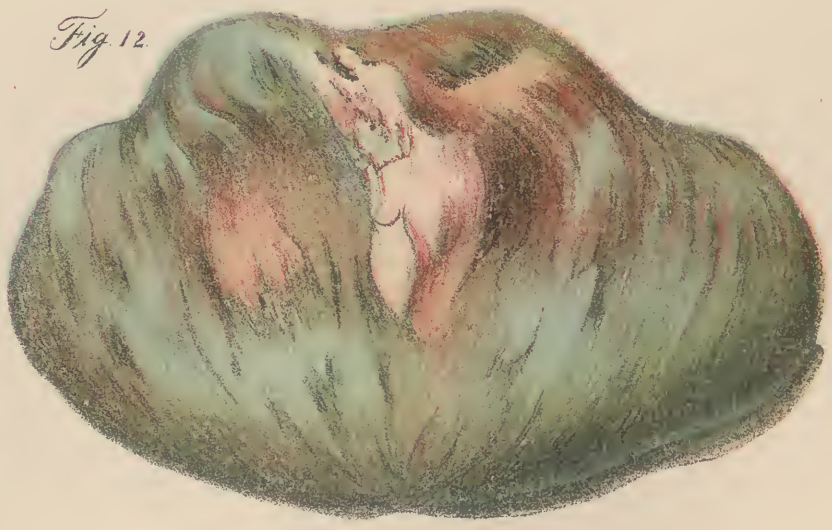


Fig. 10.

Fig. 11.

Fig. 12.



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Sec. III. *Tab. XI.*

INTERLOBULAR EMPHYSEMA.

VESICULAR EMPHYSEMA.

Fig 1.

Fig 3

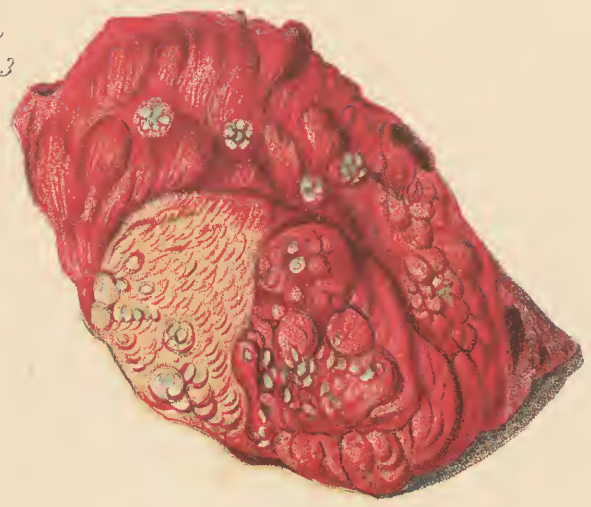

Fig. 2.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.

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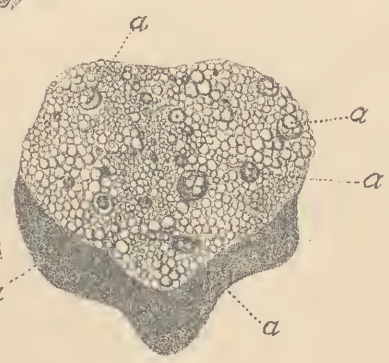

Fig. 9.

Fig. 10.

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TABLE XI.

FIGS. 1, 2.—*Interlobular pulmonic emphysema produced by violent expiratory efforts in coughing.*

CASE.—Man 76 years old.

History.—Affected with atheroma of the large arteries and aortic valves. Died of apoplexy.

Fig. 1 represents the apex of one lung, and Fig. 2 a portion of the middle lobe of the other, in a state of interlobular emphysema. There were enormous dilatations of whole groups of lobules and destruction of air-cells. A part of the pulmonic tissue was indurated.

Fig. 3. *Vesicular emphysema* of a lung of a woman of 49 years. She was attacked two years before her death with paraplegia of both lower limbs, from which she had partly recovered. Died suddenly.

Post Mortem.—There was found a large calcareous mass lodged between the inner and middle coats of the aorta, which was in a state of partial aneurism from its origin to the end of the arch. The inner coat was atheromatous. Outside, the heart was covered with very thick layers of fat; the organ was softened. The lungs partly red and partly grayish, pressed forward on opening the thoracic cavity. Larger and smaller sacs filled with air projected from its surface, and lifted the pleura in many places. When one sac was opened, the others, communicating with it, collapsed. The lungs were lighter than usual, and the bronchi contained but very little serum. The liver was infiltrated with fat.

Fig. 4. Vesicular emphysema in the lungs of a 50-year old woman. Suffered for years from bronchial asthma, with extremely violent cough, dyspnœa, and sense of suffocation. Tympanitic sound and bronchophony on both sides of chest, in front; purulent sputa. Two months before death attacks of vertigo and syncope, repeatedly. Died of asphyxia.

Post Mortem.—Sudden protrusion of lungs from opened thorax. The dilated cells formed groups of bluish and reddish color immediately beneath the pleuræ, and had a generally congested appearance.

The bronchi were all softened and filled with mucous and pus. In many places they were dilated. The blood in the heart was liquid (as usual in asphyxia); the liver strongly infiltrated with fat. Other organs normal.

Fig. 5. Two air-cells enlarged and thickened. Fig. 7, a largely dilated air-vesicle, opened at (a) showing its inside lined with a vascular layer. Fig. 8, dilated air-cells, natural size. Fig. 6, the same, magnified, showing coalescence of the alveoli and infundibula, and the broken down septa. (A. Figs. 6, 8, orifices of dilated bronchi.) (B. Figs. 7, 9, destroyed septa.)

Fig. 10, a pseudo membranous cast of trachea and bronchi expectorated by a child affected with pseudo membranous angina. (t. Trachea.) (*b. Bronchi.*)

are never infected from them, whilst others are remarkably easy to be infected.

The experiments of inoculation with tuberculous matter has been carried on in a variety of ways. Some inoculate into the skin, others the abdominal cavity, the eye, the cavities of the joints; or feed the animals with food tainted with tuberculous substances, or such as contain caseous matter, etc. Atomized sputa mixed with watery or other vapors served to saturate the air to be inhaled by the animals. (*Villemin, Gaz. hebdom.* 1855, *No.* 50. *Compt. rend. LXI.* 1866, *and Etudes sur la Tuberculose*, 1868.)

(*Lebert Bulletin de l'Academie XXXII; Gaz. Medic. de Paris*, 1867, *Nos.* 25–29; *Lebert and Wyss: Virch. Arch.* 40 *Vol.; Waldenberg, die Tuberculose*, 1869; *Langhans: die Uebertragung*, 1868; *Klebs: Wirch. Arch. Vols.* 44, 49. *Arch. fuer exper. Patholog. Vol.* ,1 *Tagblatt der Naturforscher*, 1877; *Conheim and Fraenkel: Virch. Arch.* 45 *Vol.; Tappeiner, Lyppl, Schweninger: Tagblatt der Naturforscher*, 1877; *Tappeiner: Virch. Arch. Vol.* 74; *Orth: Virch. Arch. Vol.* 76; *Bollinger: Arch. f. exper. Patholog. Vol. I.*)

However true it be that the anatomical mark of Tuberculosis lies in the presence of nodules in certain organs and tissues, yet not every nodular form nor every cellular congerie, although morbidly formed, is equivalent to true tubercles. For instance, when all sorts of substances (even the most harmless) be introduced into the tissues through the blood or otherwise, provided they are in a high state of division, they will produce, where they are deposited, a cellular infiltration, anatomically resembling tubercles. The clinical conditions will, however, be either different from those connected with tuberculosis, or inflammation and suppuration will at once mark it as a distinctly non-tuberculous disease. Even spontaneously generated, tuberculous or rather nodular forms, such as are found in cutaneous lupus, or in the serous membranes diffused here and there, cannot be considered tubercular.

There are a number of conditions in the body where inflammatory granulations are produced, which are to be counted as tubercular, although tubercles be absent; for not only are the clinical course of the process and the development of the inflammatory neoplasm indicative of tubercular peculiarities, but also eventually tubercles develop in those granulations. Such a condition in an organ like the lung very often obscures the diagnosis, for simple inflammatory processes often bear the same progressively destructive character as tuberculosis of the organ. As a rule, true tuberculosis manifests itself clinically and anatomically alike.

Etiology of Tuberculosis.

After the brilliant and much promising discovery of the *Bacillus Tuberculosis* by Robert Koch, and the manifold confirmations of the actual existence of the fungus in tubercles and kindred morbid formations, it was reasonable to expect the question of *Tubercular Etiology* settled; yet such is not the case. In the latest medical literary publications quite a passionate war of words is carried on between the schools of Berlin and Vienna, on the very subject of the bacillus, which threatens to unsettle the whole question once more. Until the right of the one or the other side in the controversy is decided by further research, it is necessary to hold on to such facts as are beyond cavil. The cardinal facts about tubercular etiology are:

1. Real tubercles do not develop spontaneously, but are products of reactive processes carried on by the body to overcome the effects of infectious or contagious bodies of a special kind.

2. No substances or bodies, except micro-organisms, are able to produce in the body and to call forth therein such definite morbid phenomena and specific morbid anatomical products as are found in tubercular conditions of the tissues.

3. That the tubercle itself, and almost all of its transformations, provided these contain the specific virus proper to them, are capable of reproducing the phenomena peculiar to tubercle, when introduced into other parts of the same individual or into others.

Whether the tubercular bacillus constitutes a parasitic fungus *sui generis*, as R. Koch and his adherents claim, or not, is really immaterial. The fact is sufficient that tuberculosis is an infectious disease of a parasitory origin

Very important is the question, whether other forms of phthisis pulmonalis resulting, for instance, from caseous pneumonia and broncho-pneumonia, scrofula, and kindred pulmonary lesions, owe also their origin to the same bacillus or not.

If, as R. Koch states (*in Etiologie der Tuberculose, Berlin, Klin. Wochensch., und uber Tuberculose; Arch. f. Anatom. & Physiolog., p.* 190), the bacillus can only exist in a temperature of from 30° C. to 42° C., and is necessarily developed in the animal body and can scarcely develop outside, what is the etiology of tuberculosis derived from scrofula or rather developed from inequality of venous circulation in the lungs and bronchi, with irregular respiration, inadequate nutrition, cellular emigration, and partly œdomatous infiltration into the tissues; in fact, that condition of the body described as scrophulosis? All of these morbid manifestations are produced by *faulty hygiene*, in the animal and human body. Practically, the remote cause is of lesser importance than the immediate, and here the clinical phenomena can lead far easier to the solution of the problem of tubercular etiology than the anatomical.

General Physical Symptoms of Broncho-Pneumonia, Bronchitis, and the several forms of Pneumonia.

1. Diseases of the bronchi, even after they have existed for some time and have become a source of anxiety and annoyance to the patient, may still be very difficult to diagnose. When, for instance, the seat of the bronchial trouble be at their origin and close to the place of their bifurcation, there will be violent cough, a constant sensation of tickling, and soreness in the lungs, without any other objective signs. The same is the case when the disease is situated in the middle of the lung and surrounded on all sides by parenchyma containing air. In such cases, only the sputa would positively indicate, to a certain extent, the nature of the lesion. Patients will sometimes expectorate large quantities of sputa without manifesting any other physical symptoms, as Wintrich has noticed it. Here, too, only the sputa will indicate the existence and the nature of the lesion. Constant absence of tissue-elements of the lung in the expectorate points to a bronchial disease. When the expectorate is very copious, but comes at long intervals, dilatation of the bronchi will be indicated; when it has a bad odor, and contains mycotic bronchial coagula, especially if it is inclined to separate in layers, putrid bronchitis may be well presumed to exist.

In peripheral and readily recognizable alterations of the bronchi are conditions which lead to enormous accumulations of liquid in their cavities, and give rise to *rales*, or moist rattling. According to the viscid condition of the liquid are rales different. Very viscid liquids give rise to dry rales or *rhonchus*, which, if they exist in the larger bronchi, produce a snoring; if in the finer bronchi, produce a whistling or sibilating rhonchus. Where the secretion is not at all viscid and more flowing, moist rales or mucous rattles are produced. The wider the bronchi the coarser will the vesicular rales be.

Where there is much secretion accumulated in the smaller and smallest bronchi, the sound will be of the crepitating, crackling kind.

The *intensity* of the rale will indicate the seat of the disease, its extent, the diffusion of the disease process. In alterations existing only in the bronchi no click or metallic ring exists; this always predicates a condensation or induration of the adjacent lung-tissue, recognizable by a dull, or dull-tympanitic resonance. Whether the accumulated liquid in the bronchial cavity be purulent, mucous, or bloody, the sputa alone can decide.

2. When large quantities of liquids or secretions are so accumulated in the bronchi as to close them up the physical symptoms will soon indicate it, only it must not always be taken for granted that an obstructed bronchus is always filled with liquid. It might

TABLE XII.

FIGS. 1, 2, 3, 4.—*Chronic catarrhal inflammation of the larynx, trachea, and bronchi.*

Fig. 1 shows the anterior wall of the trachea which is opened posteriorly, the thyroid body (C. T.) enormously enlarged, surrounding and compressing the whole anterior and lateral portions of the trachea, only the membranous part being free; the internal surface of the larynx and trachea in a state of hyperplastic inflammation. Neoplastic deposits are lodged upon the mucous membrane, partly in patches (P. S.), partly in spheroid nodules, forming a somewhat coherent membrane (P. S. T.), which extends down the bronchus and into some of its primary divisions. To the right, the carotid artery (A. C.) and the jugular vein (V. I.), the latter containing purulent coagula, cause complete obliteration of the circulation.

Fig. 2 shows the nodular deposits extending into the dilated bronchi, one of which terminates in a cavity of a tubercular lung (P. P.). The base of the right arytenoid cartilage is exposed by insertion of a hook, and shows loss of substance produced by ulceration. Fig. 3 shows bronchial bifurcation, each division being alike affected by inflammation. (P. P.) enlarged papillæ.

Figs. 4, 5. *Acute pharyngo-laryngitis and epiglottitis.*

CASE.—L. W., a man 28 years old.

History and Symptoms.—Affected with a light form of angina of the fauces and larynx. Second day after the attack exacerbation, and taken with severe acute gastritis; dyspnœa set in. On the fifth day purulent spots found on both sides of the pharynx. Respiration a little easier. The following night, delirious. Sixth day, decided typhoid symptoms; very dry skin; hot, dry tongue. Pulse very frequent and feeble. Stertorous breathing; coma. Died the eighth day.

Post Mortem.—Brain anæmic. Serum in arachnoid cavity. Heart and lungs nearly normal. Nothing remarkable about the bronchi. The epiglottis (Fig. 1) very red and much swollen, and raised high up in the pharynx above the level of the tongue. All the soft parts between the epiglottis, the root of the tongue, and upper edge of the larynx enormously swollen. The epiglottal edges rolled in and formed a groove (a) leading to the vocal orifice. Situated behind and on each side of the glottis were two very thickly swollen and very red sacs pressing upon it and nearly closing it up. They extended from the external and posterior portion of the pharynx to two inches below the upper part of the œsophagus. On opening, the sacs were found to contain blood and pus. The folds of the mucous membrane were infiltrated with serum. Fig 4 shows the fauces and the visible portions of the pharynx very red and swollen; the epiglottis high up in the cavity. Fig. 5, a longitudinal section of the glottal cavity.

(A.) tongue. (B.) epiglottis. (C.) cricoid cartilage divided. (D.) epiglottal folds. (E.) inner surface of the anterior wall of the larynx. (F.) inner surface of same cavity below the vocal cords.

be closed by all sorts of solid substances coming from without or secreted internally. Compression of the bronchi often produces similar sound-phenomena.

3. Closed bronchi will show on inspection that they do not partake in the respiratory process, by retraction of the thoracic wall during inspiration. When the larger bronchi are occluded, or, when very many smaller ones are so, there will be *cyanosis* of the skin, and dyspnœa will indicate the obstacle to respiration. Vocal fremitus will be absolutely wanting in the region of the occluded bronchi; as long as the air-cells contain air there will be no dullness on percussion. When the obturation lasts for a considerable time the sound will be tympanitic, due to absorption of portions of the stagnant air behind the obstructed portion.

4. There is no respiratory murmur in the diseased region audible by auscultation, because the respiratory murmur cannot be continued beyond the obstructed portion. Nor will there be any bronchophony. Very copious mucous rales will point to great accumulations in the cavity.

Foreign bodies, if they penetrate and obstruct the bronchi, will have to be ascertained. If fibrous coagula are the cause, a portion of fibrin is always found in the expectorate. Compressions have to be ascertained by careful examination of the heart, vessels, etc.

5. Between perfect closure of the bronchi and their entire freedom stands the constriction or stenosis of the bronchial cavity. The more constricted the more will the symptoms be like those of occlusion.

When the constriction is not very great it will be manifested by increased strength of the respiratory murmur. Occasionally the expiratory act is longer than usual and is accompanied by snoring, sibilating crepitation.

In progressive stricture vocal fremitus and bronchophony grow feebler, the respiratory movement and the respiratory murmur lose their force, whilst the inspiratory act will be characterized by retraction.

6. Dilatation of the bronchi can only be diagnosed when it takes place in spots and is widely diffused. Only very copious expectoration will indicate such a condition.

Circumscribed bronchiectases are manifested by nearly the same physical symptoms as cavities. Differential diagnosis between excessive broncho-ectases and cavities the sputa alone can determine, as mentioned above. Examination of the expectorate for shreds of pulmonic tissues has to be carried on for some time; for in the primary conditions of lung cavities those shreds are often wanting. The history of the case will often throw light upon the subject and help to make out a diagnosis.

Tympanitic sound by percussion on the thorax in bronchial diseases is only then produced when the bronchi are partly closed up and leading to softening of the lung-tissue; or when they are very much dilated and situated very superficially. When they are surrounded by portions of the lung having a thickness of at least five centimetres no tympanitic sound will be heard. It is especially necessary to percuss over the bronchi more forcibly than over other portions of the thorax, in order to elicit the bronchial sound through the lung-tissue. Bronchi situated in a depth of at least five centimeters, even if they be very much dilated, will not yield any tympanitic sound. As dilated bronchi usually contain much secretion it often happens that the sound becomes masked when they are filled, and reappears when they are emptied by expectoration or otherwise.

As a rule bronchiectasis exists in the posterior and lower portions of the lungs.

Only superficially situated portions of the lung are accessible to diagnosis by auscultation and percussion. Centrally located diseased parts can only be recognized by the expectorates; thus do rusty-colored sputa indicate fibrinous pneumonia. Centrally located gangrene and abscess are diagnosed by dark or greenish-colored expectorates.

A very important part of diagnosis forms the exact tracing of the pulmonic boundaries, for they may deviate in several ways from the normal; first, by irregularity on both sides; second, both borders passing their limits; third, by displacement downward.

A.—*Unequal position is oftenest found of the apices of the lungs in the course of chronic induration or shrinking of the tissues, and forms a very important symptom in slowly developing pulmonary phthisis.*

B.—*Surpassing the normal boundaries by any lung, in any direction, is very significant of a condition of alveolar emphysema.*

Such a condition may exist in one or in both lungs. If in the right lung alone, it is best recognized by *its lower border* passing its normal limits.

If in the left lung, its increased volume will not only make it pass the normal downward, but its *anterior median border* will *approach the left sternal border*, and the lung will cover the pericardium, anteriorly, far more than usual, so that the region of cardiac dull sound will be diminished in size, or will altogether disappear. A greatly enlarged volume of the lung must necessarily depress the diaphragm, the apex-beat will then appear lower, say in about the sixth intercostal space, and the locality below the heart, which is characterized by tympanitic sound, will proportionately lose in extent. Increase of volume of a lung, if existing for some time, often changes the configuration of parts of, or of the whole chest.

C.—In very severe derangements of the lungs vocal fremitus and bronchial resonances lose very much of their force, the same also with the respiratory murmur; the weakness is due to much loss of force of the respiratory movement.

Decrease of volume of the lungs may take place under two circumstances. One, by compression, from below upward, by any distension of the abdominal contents, from whatever cause. Two, by shrinking and atrophy of the parenchyma itself; also by compression by liquid or solid foreign substances in the thorax. Atrophy of the lungs is manifested by the following physical symptoms: Inspection and percussion of the thorax will show a feeble circumference of the chest cavity, narrow intercostal spaces, feeble respiratory movement, scoliotic curvatures of the spine. Percussion will show the lower borders of the lung to stand higher than usual, whilst the healthy lung will, in the same ratio, reach much lower down. Accordingly, when the left lung is atrophied the apex-beat will be found in the fourth intercostal space, whilst the subcardiac region will be marked anteriorly by a wider tympanitic-sound region. When the median border of the left lung is drawn outward the origin of the pulmonary artery becomes more exposed, and its systolic filling readily visible and palpable in the second left intercostal space. Auscultation of such a lung will show the same characteristic sound as that of an indurated lung. There will also be increased vocal fremitus, bronchophony and bronchial respiration. During each inspiration the lung normally increases in volume, especially when deep breathing is carried on, and the complementary spaces of the thorax are filled out. When inflammatory processes have produced adhesion, and the complementary spaces are thus obliterated, the respiratory displacement of the borders of the lungs will be either very limited or cease altogether. The liver will retain its position in both phases of the respiratory act, and the cardiac dull-sound region and subcardiac tympanitic region will remain normal.

D.—Liquids collected in the alveoli of the lungs manifest physical symptoms different when they contain air-bubbles than when they contain none.

In the first case—which is really the most frequent—auscultation will discover *crepitating rales*. Percussion will usually produce tympanitic deep resonance, whilst inspection will show disturbed respiratory motion. The quality of the liquid (whether it be blood, serum, pus, etc.) must be ascertained by the sputum. Very often the history of the course of the case throws light upon the subject, and the nature of the liquid surmised. One fact must not be

DISEASES OF THE ORGANS OF RESPIRATION.

CATARRHAL INFLAMMATION OF LARYNX,

TRACHEA AND BRONCHI.

Fig. 1.

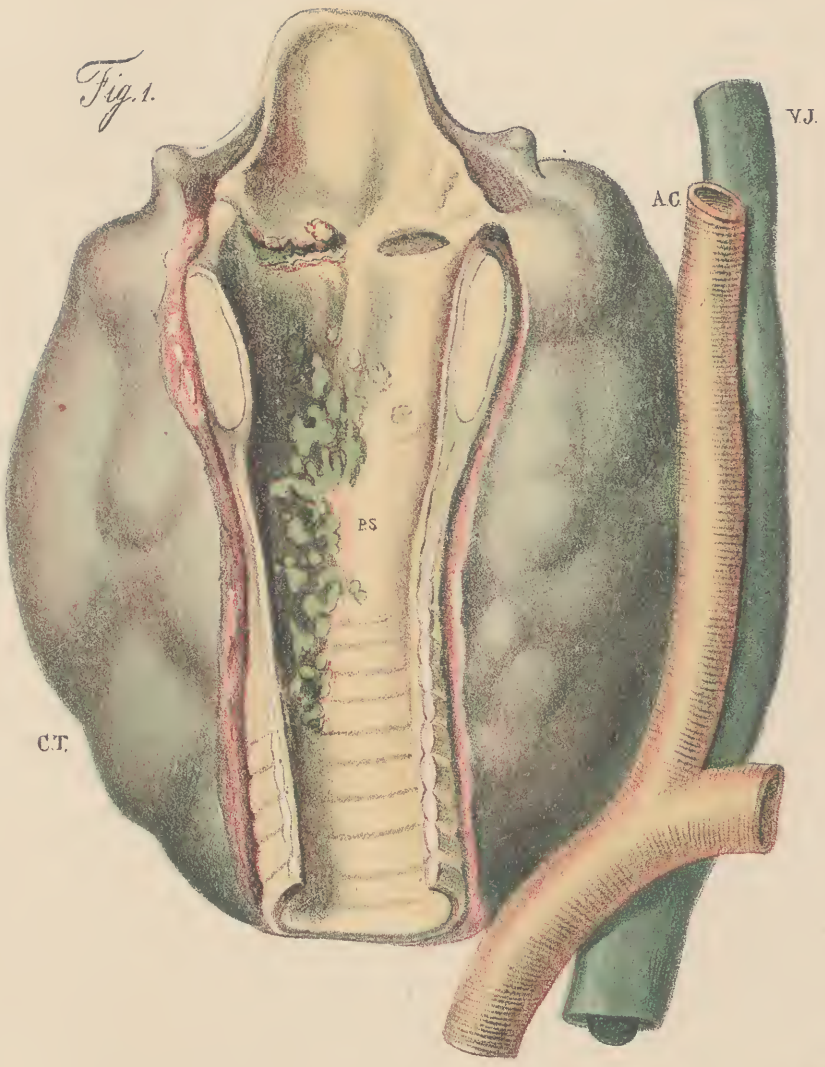


Fig. 4.

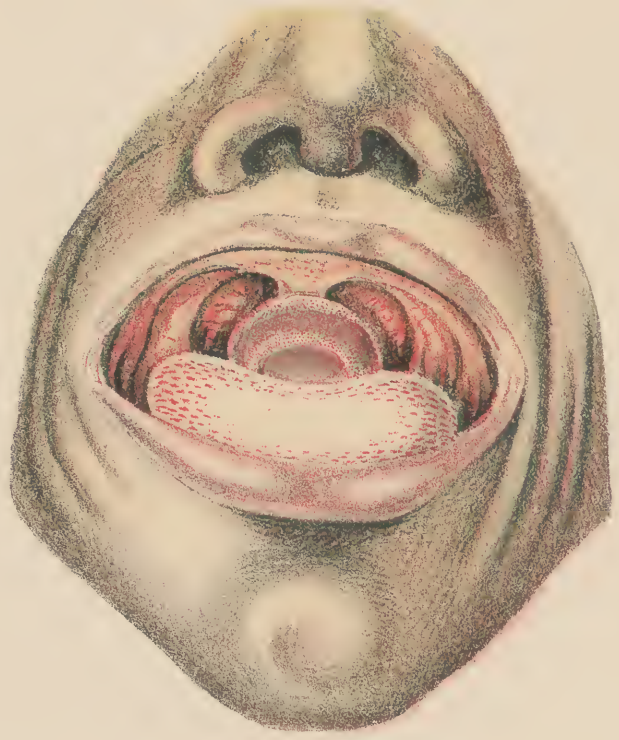


Fig. 5.

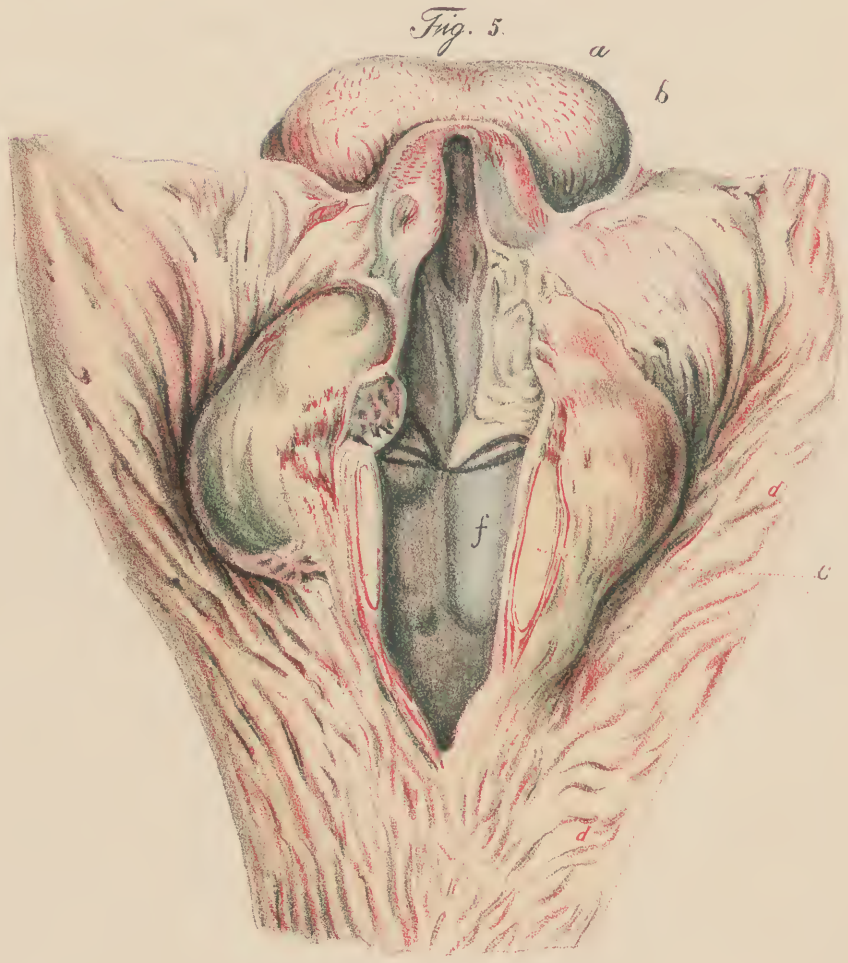


Fig. 2

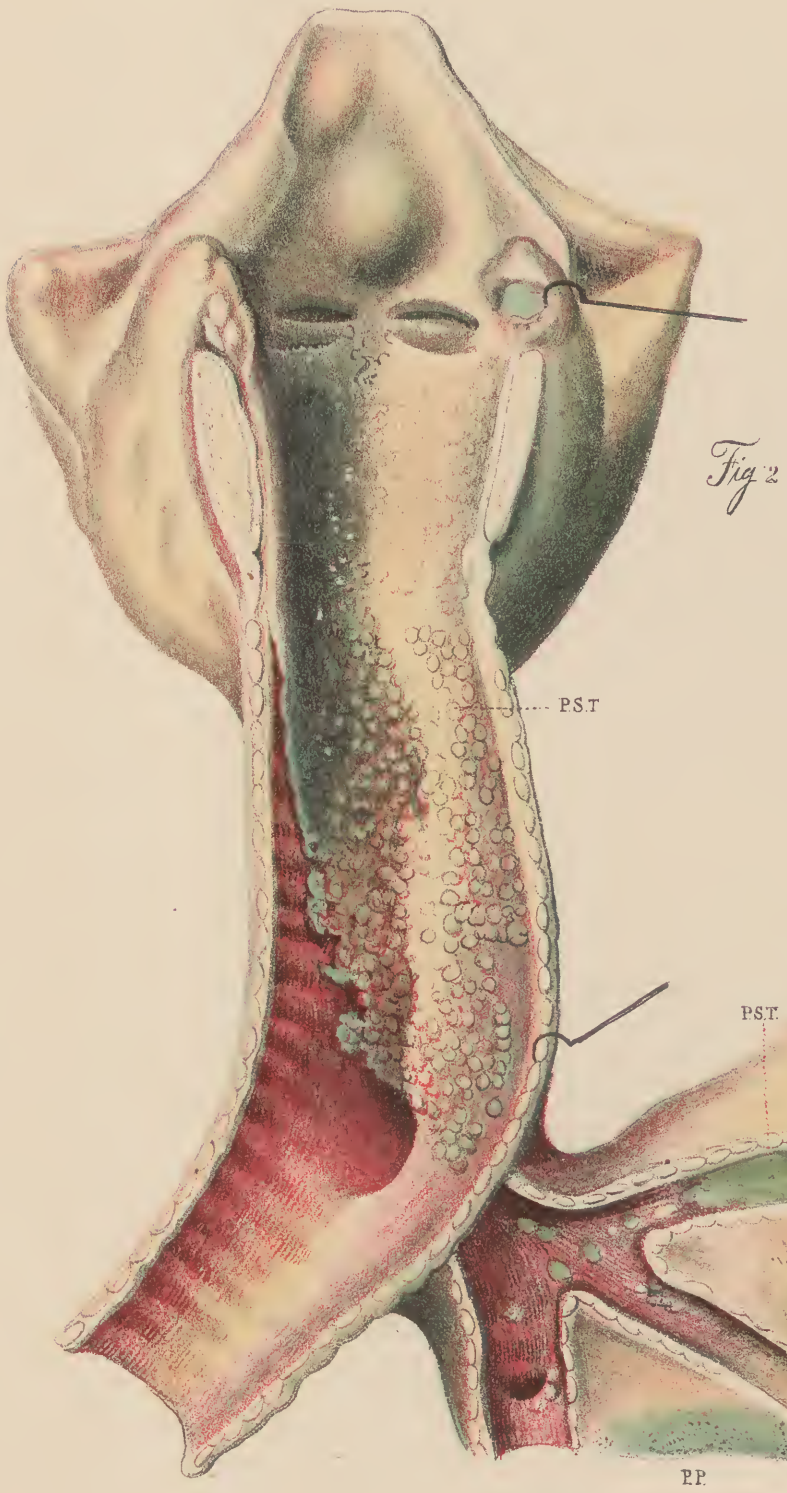
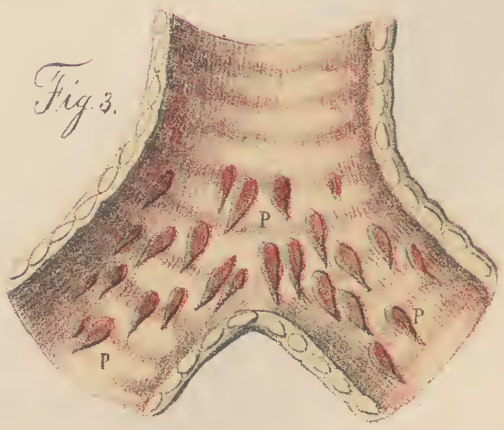


Fig. 3.



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Sec. III *Tab. XIII.*

ACUTE LARYNGITIS.

PHLEGMONOUS LARYNGITIS.

Fig. 1

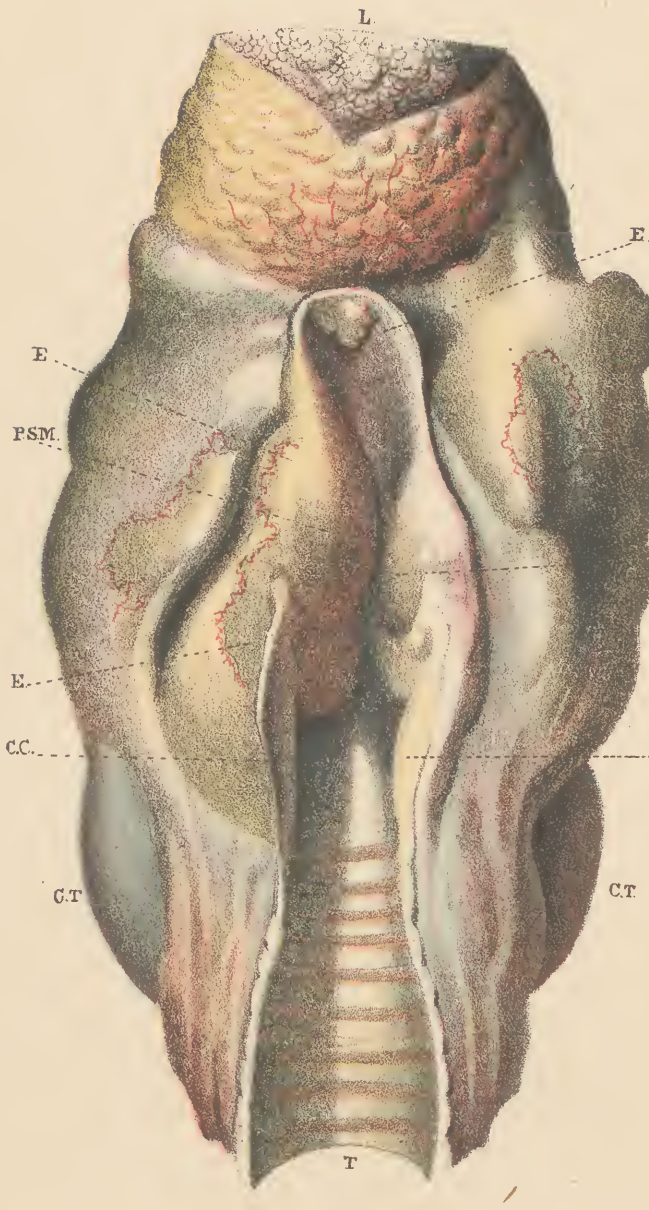

Fig 2

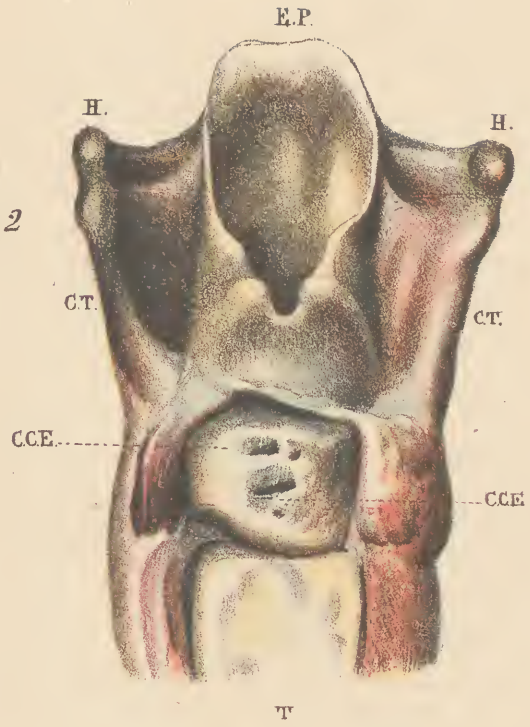

Fig. 2.

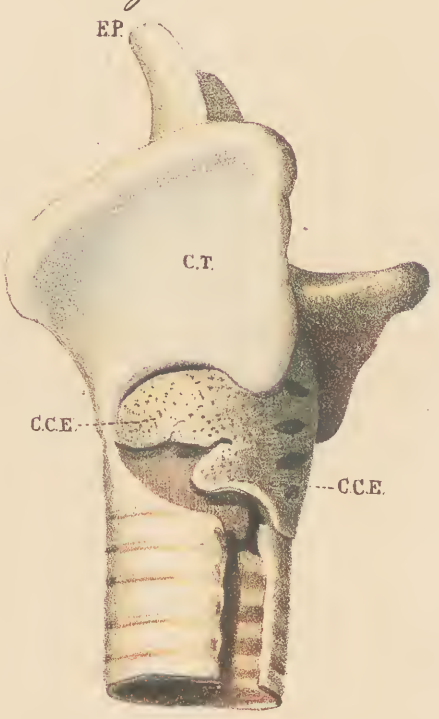

Fig. 3.

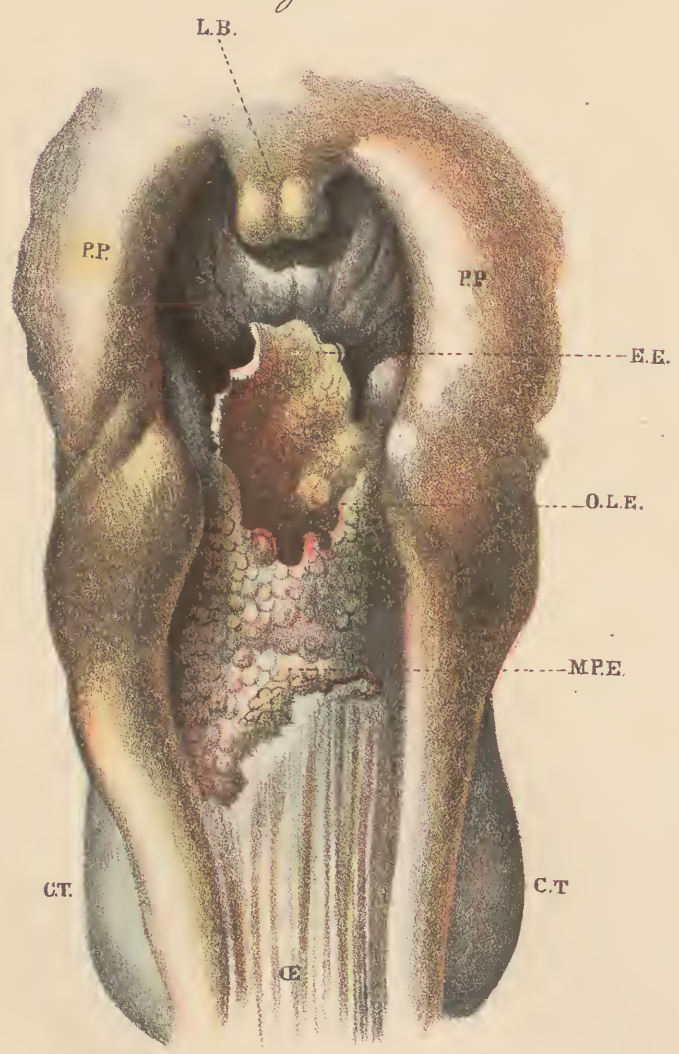

Fig. 3.

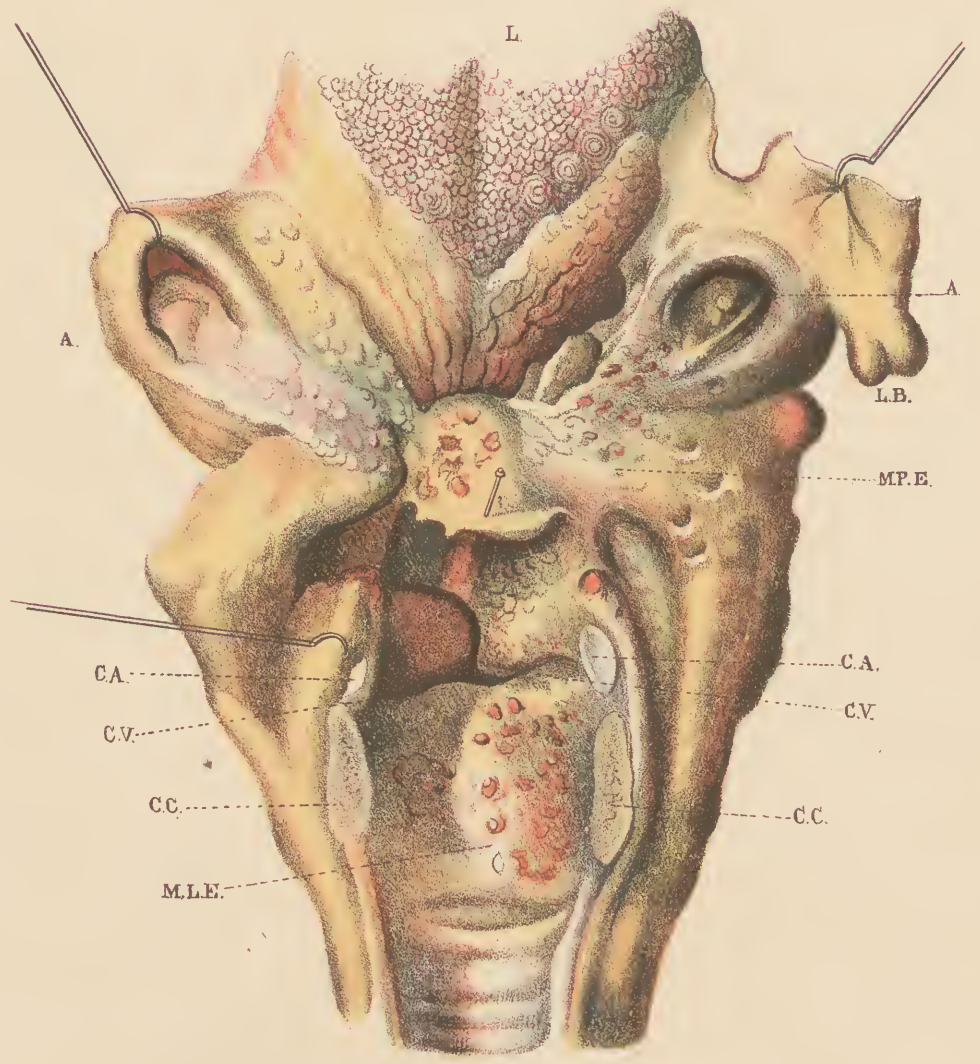

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TABLE XIII.

Fig. 1. *Acute laryngitis with œdematous infiltration into the sub-mucous tissue in the epiglottal region.*

Case.—V. P., 50 years of age inveterate drinker.

History.—Taken with general weakness and sense of fatigue in the limbs. *Symptoms.*—First three days pain in limbs and in throat; frequent pulse, burning hot skin, deep red mucous membrane of the mouth and pharynx; swelling of the uvula; is unable to swallow; is short of breath. Since the fourth day very hurried breathing, nearly suffocates, cannot speak above a whisper. Soft palate very red and more swollen; anterior pillars so much enlarged as to close up the throat. Fifth day, much worse, excessive pain in throat, is threatened with asphyxia. Dies the following night. *Post Mortem.*—Ary-epiglottal folds, the mucous membrane investing the whole posterior and lateral regions of the larynx, portions of the pharynx, and the anterior and superior borders of the epiglottis presented a whitish yellow appearance, and infiltrated with pus. The mucous folds (P. S. M.) were enormously tumefied and touched each other; only posteriorly was a narrow passage left for the air. The glottis and sub-glottal parts were nearly normal. The infiltrate was diffused all over the whole mucous and sub-mucous tissues. A number of erosions (E. E.) existed. The tonsils were sound. The anterior surface of the epiglottis was covered with scabs, and its inferior portion driven backward and helped to close up the glottis.

Figs. 2, 2^1. *Phlegmonous laryngitis and formation of abscesses in the perichondrium.*

Case.—A young man of 25 years. Was taken to the hospital in a dying state. No history. *Post Mortem.*—The posterior surface of the larynx presented a ridge along the cricoid cartilage, which was corroded, denuded, and perforated. In a number of places it was reduced to a very thin shell. The mucous membrane, which was thickened, formed a sack, and was filled with a great quantity of pus. The perichondrium was obliterated. The arytenoid cartilages were surrounded with a thickened membrane and the crico-arytenoid articulations destroyed. The muscles infiltrated. The necrosis and erosions extended from behind forward on each side. Fig. 2^1 represents the left lateral surface of the cricoid. (C E E, erosions.) (C T, thyroid cartilage.)

Figs. 3, 3^1 present the posterior surface of the larynx of an individual who died of laryngeal phthisis. The bifide uvula (L. B.) and the posterior pillars of the velum palati (P. P.) were much thickened, the epiglottic edges corroded and beveled (E E). The upper laryngeal edge and the mucous membrane investing its posterior surface (M. P. E.) were dense, swollen, corroded, ulcerated, and covered with carunculous eminences. Fig. 3^1 presents the arterior surface of the epiglottis (M. P. E.) in a similar condition as the other, and extending to the base of the tongue. The opened larynx shows the alterations on its surface. The base of the right arytenoid cartilage (C. A.) was denuded, corroded, and partly ossified. The lungs and the lymph-glands in the thorax were filled with tubercles. (A., arytenoid cartilage, the mucous membrane ulcerated and thrown back; C. A., arytenoid cartilage itself; C. V., vocal cords; M. L. E., erosions on mucous surface.)

forgotten, that is, that mucous rales do not *always* indicate a disease of the alveoli, for liquids collected in the finer bronchi will produce the same symptoms. When the alveoli are filled with caseous or fibroid masses containing no air, the following symptoms will be manifested: total or partial immobility of the chest during respiration; vocal fremitus and bronchophony increased in intensity over the diseased portion, ægophony; dull sound on percussion, gradually passing into *tracheal sound of Williams*, over the upper lobes. Bronchial sound, ringing or metallic sounds, ringing rales heard by auscultation. The sputa will here also form the most reliable means of diagnosis.

When circumscribed cavities are situated beneath the thoracic wall, no matter whether they are formed by dilatations of bronchi, loss of pulmonic tissue, or partial pneumo-thorax, the same tone will be obtained by percussion. Of course, the sputa will have to be examined to ascertain, from the nature of the detritus they contain, whether the bronchial tissue or the pulmonic is undergoing destruction. The most definite symptom of the presence of a cavity is a metallic sound heard on percussion. As this kind of sound is also heard in other lesions, other symptoms must be noticed in connection with this, and these are: diminished visible respiratory motion of the part of the chest over the cavity, collapse of that portion to a greater extent, sudden protrusion of some intercostal spaces during forcible expiration (as in coughing, etc.), weaker pectoral fremitus; increased bronchophony, occasionally ægophony, and when the cavity is extensive and its walls are smooth, metallic sounds, associated with the others, are heard by auscultation. Usually muffled tympanitic, seldom metallic, sounds are produced by percussion. When a cavity is totally filled with liquid its tympanitic sound will disappear; when partly filled with liquid and partly with air, especially when the liquid freely moves in the cavity, the tympanitic sound will sometimes disappear and reappear as the person moves. The so-called sound of *a cracked pot* (*pot fele*) is often produced by percussion over cavities. Auscultation will discover bronchial respiratory murmur. In large, smooth-walled cavities amphoric resonance is often produced. The rales are all of the ringing kind. When they are very intense their vibrations are frequently plainly palpable externally.

To the cervical portion of the respiratory organs belong the larynx, trachea, and the thyroid body; for all three constitute one apparatus even in the earliest fœtal period. (*V. Baer, Remak, Koelliker.*)

Besides many congenital anomalies which may exist in either of the parts of that apparatus, a number of morbid alterations may take place in the organ, some of which cause local, and some general, disturbance of the respiratory functions. The larynx may be dislocated by mechanical injuries, or pathological changes of its own tissues, or others adjacent to it. Its form and volume may undergo greater or lesser modifications. It may become dilated or contracted beyond the normal, or may be invaded by living or lifeless foreign bodies, which would affect its tissue, its form, or its volume.

Morbid Changes in the Laryngeal Mucous Membrane.

In jaundice, and in a state of infiltration of the bodily tissues with hæmatine, the laryngeal mucous membrane assumes a more or less deep yellow color; especially the epiglottis, the false vocal cords, and the ary-epiglottal folds become thus intensely colored. The different portions of the mucous membrane being attached to the laryngeal walls, in some places very loosely, in some firmly, active arterial hyperæmia produces a deeper red color in the firmer parts and lighter color in the looser portions. Hyperæmia from venous stagnation is more uniformly diffused, and its color is darker all over the whole of the lining membrane than in the arterial.

Many corrosive and poisonous substances produce in this, as in other mucous membranes, more or less severe hæmorrhages. The coloration produced by it will differ according to the intensity of infiltration and quantity of diffusion in its several parts. They may form small red or dark spots or extensive ecchymoses, or very dark colored plaques. Anæmia, both general and local, makes the membrane look pale. In consequence of great tension, produced by sub-mucous inflammatory infiltration, as, for instance, in dropsy associated with Bright's disease, in general bodily marasmus, in old age and in severe cachexia, the membrane will have a peculiar dry, glistening, thin appearance. In anæmia of chronic laryngeal catarrh it will be swollen and highly infiltrated with serum.

Acute Catarrhal Laryngitis.

Catarrhal inflammation most frequently affects the mucous membrane of the larynx, and is manifested by injection, hyperæmia, swelling, softening of the membrane, and by increased as well as altered secretion. Whilst the hyperæmia is only perceptible in the looser portions, the swelling extends everywhere. The sub-mucous tissue and its glands are likewise involved. This diffuse swelling, which closes the glottis, constitutes the most important symptom of the disease. Owing to the great elasticity of the membrane rupture of vessels very seldom happens in acute catarrh, so much oftener is there loss of epithelial tissue (excoriations). Ecchymoses are often formed in conjunction with excoriation. In the early stages of the inflammation the secretion is rather scant—there is only burning and dryness of the larynx; later, a tough, transparent, glassy secretion, occasionally mixed with blood, appears. When it is derived from the sub-mucous glands it comes in the form of little transparent balls or lumps, when from the folds of Morgagni it is yellow and turbid; from excoriations it appears like pus, and is very abundant. The denuded places mostly found on the vocal cords appear as bright red depressions with very sharp edges. In simple catarrh the ulcerations never penetrate the whole thickness of the membrane; but when it is formed in consequence of primary tuberculous, syphilitic, leprous, or lupous ulceration, then the whole thickness of the membrane is penetrated. The intensity and extent of the catarrhal inflammatory process varies very much. The epiglottis, the ary-epiglottal ligaments, the vocal cords may be singly affected, or the whole extent of the larynx may become involved. The symptoms will be according to the intensity and the extent of the inflammation. When the catarrhal process extends from the trachea upward there will be dyspnœa and vocal derangement; when from the pharynx downward, it will be marked by difficulty in swallowing.

Acute laryngeal catarrh is usually a transitory disease, ending, as a rule, in recovery. Fatal terminations have been reported by Porter, Ruhle, and Tobold. Spasmodic or pseudo-croup has been considered by some authors as a form of laryngeal catarrh; by others it is thought to be merely a neurosis, due to reflex action brought about by irritation of the membrane, by accumulations of irritant mucus, or other secretions in the laryngeal cavity.

The etiology of laryngeal acute catarrh was, and still is, considered by many to be cold, or a sudden change from a very high to a very low temperature. Its contagious nature can by no means be explained by mere cold-taking, as it is well known that persons affected with laryngeal catarrh will communicate it to others who were never exposed to change of temperature. Klebs (*in Arch. f., Experiment. Pathol., Vol.* IV) has called attention to the constant presence of micro-organisms in the secretions of persons thus affected. These organisms, which are always present in all buccal and laryngeal secretions, may become the carriers of the contagious substances which may develop either by their agency or by others in the secretions vitiated by the morbid process. Lebert (*in Berlin. Klin. Wochenschrift*) and Wirth (*in Staub Inhalation*) have, by a great number of statistics, set forth that finely-divided chemical and mechanical irritant substances are capable of producing in the respiratory passages at first catarrh of the pharynx and trachea, and then extending to the larynx swelling and ulceration of its mucous membrane. Infectious diseases, such as measles, scarlatina, small-pox, cholera-typhoid, are always associated with acute laryngeal catarrh. In measles and scarlet fever this forms one of the most important symptoms. In measles the catarrh is manifested by intense redness and injection of the larynx. In scarlet fever it assumes a desquamative form in the shape of a white-layered exfoliation of the epithelium. In hay fever, and in the latter stages of very severe cases of whooping cough, a very annoying, and often dangerous, form of laryngeal catarrh is developed. Letzerich (*in Virchow's Arch., Vols.* 49, 60), as well as Henke (*in Arch. f. Klin. Mediz.*), has discovered micro-organisms

TABLE XIV.

FIGS. 1, 2.—*Osteo-chondroma of Larynx.*

Case.—A. W. Carpenter, 40 years old.

History and Symptoms.—Entered Charity Hospital, Berlin, April 1,18—.—The very emaciated patient had suffered for a long time from harassing cough and enormous expectoration. Was moribund when brought to hospital. Was in a complete state of coma. Died an hour later. *Autopsy.*—Twelve hours after death. In cranial and thoracic cavities all viscera normal except the larger bronchi, which contained some greenish-yellow secreta. The mucous membrane of trachea was very red and very soft. After dividing the posterior pharyngeal wall the glottis (Fig. 1) was found extremely narrow, the left half of the larynx very much swollen, although of normal color, and its mucous membrane but little redder than usual. The stricture of the glottis was caused by an osteo-chondroma situated on thyroid cartilage, and penetrating into the larynx. The intervening soft tissues were partly compressed and partly altered by ulceration and gangrene.

Fig. 1 presents the pharynx and œsophagus divided and their walls thrown back. *A*, root of the tongue; *b*, posterior surface of epiglottis; *c*, larynx, swollen on the left side; *d*, apex of posterior cornu of hyoid bone; *e*, commencement of œsophagus.

Fig 2.—*The whole of the right half of the larynx is left entire; the left is divided in the middle.* *A*, root of tongue; *b*, epiglottis; *c*, osteo-chondromatous tumor of thyroid cartilage; *d*, larger cornu of hyoid bone; *e*, upper portion of the left half of the thyroid cartilage; *f*, opening for passage of superior laryngeal artery; *g*, lateral part of cricoid cartilage surrounded by the tumor; *i*, border of mucous membrane; *k*, left sterno-hyoid muscle; *l*, left sterno-thyroid muscle; *m*, crico-thyroid muscle; *n*, thyroid gland, slightly indurated; *o*, inner surface of sup. thyroid artery, atheromatous; *p*, tracheal cavity, normal; *q*, portions of pharynx and œsophagus.

Figs. 3, 4.—*Perichondritis of Cricoid Cartilage with Formation of Abscess.*

Case.—A. Harknow, man of forty years, admitted into Charity Hospital, Berlin, Feb. 12, 18—.—Had severe pain in the larynx, voice deep and hoarse, stridulous respiration, croupy cough, expectorates muco-pus. Pulse very rapid and soft. Face pale. 19th of February, dyspnœa and sense of suffocation, orthopnœa, face livid. Performed laryngotomy; did not relieve him; died the following night. *Post Mortem.*—Abdominal, cranial, and most of thoracic viscera normal. Slight swelling in lower laryngeal region externally. Internally, epiglottis normal; glottis of normal color but much constricted. Posteriorly, the mucous membrane between the arytenoid cartilages, thick and slightly red, excoriated. In lower portion of larynx a soft tumor covered by blue-colored mucous membrane. Beneath it an abscess of an inch in width, in which the cricoid cartilage was situated, perfectly isolated from its surrounding soft tissues. The abscess extended on both sides forward and downward. On further dissection the cartilage was found to be attached only above and in front; behind it was completely surrounded by the abscess. The whole was denuded of its perichondrium, in many places corroded. The trachea and bronchi were intact.

Fig. 3.—*A*, root of tongue; *b*, epiglottis; *c*, upper edge of glottis; *d*, superior cornu of thyroid; *e*, reflected sides of pharynx and œsophagus; *f*, divided larynx, internal surface; *g*, divided cricoid in abscess cavity; *h*, cavity of the abscess; *i*, internal wound made by laryngotomy.

Fig. 4.—Same as the previous, the epiglottis removed. *A*, root of tongue; *b*, portion of mucous membrane of epiglottis; *c*, internal surface of left half of the thyroid cartilage; *d*, sub-mucous connective tissue in place of destroyed cartilage; *e*, fatty tissue beneath the epiglottis; *f*, a portion of crico-thyroid membrane still adherent to a remnant of the cartilage (f); *g*, cricoid cartilage severed by laryngotomy; *h*, internal surface of abscess; *i*, wound made by operation; *k*, *m*, sterno-hyoid and sterno-thyroid muscles; *l*, mucous membrane of œsophagus; *m*, *m*, posterior surface of thyroid.

Fig. 5.—Atrophy of posterior crico-arytenoid muscle.

Fig. 6.—Atrophy of left lateral crico-arytenoid muscle (C A L) (lateral crico-arytenoid; c t a, anteroir crico-thyroid), (C A P) posterior crico-arytenoid); (a, arytenoid cartilage.)

in the secretions and sputa of children and persons affected with these diseases.

Chronic laryngeal catarrh differs from the acute form by the nearly permanent dilation of the vessels, owing to long-continued hyperæmia. In the rare cases where the vessels return to their normal caliber they leave behind them traces in the shape of infiltration of blood pigment in their former tracks. Hypertrophy of all the layers and elements of the membrane will appear as a rough surface and rugous elevations. The hypertrophic papillæ will especially protrude upon the free surface. The secretion is either muco-pus, containing great quantities of fatty degenerated epithelium and lymphoid bodies, or serous and foamy, containing but few cellular elements. There are two varieties of the chronic catarrh corresponding to the forms of secretion. In all irritative catarrhs there is a thickening of the epithelial layer, due to extensive cell division, and great collection of lymphoid bodies, which form a thick layer of decayed structures floating in exudated serum. The mucous and sub-mucous vessels are very much, and usually, permanently, dilated, surrounded by great quantities of emigrated colorless blood corpuscles and highly hyperplastic connective tissue in a state of cell division. The glands are turgid with mucous and exfoliated epithelium. The catarrhal state caused by chronic hyperæmia and stagnation of the circulation is quite different. Only thin watery secretion, slight exfoliation of the epithelium without cell division, enormous dilatation of the vessels, sclerotic state of the connective tissue, dilatation of the lymph-spaces in that tissue, with collection of cellular elements beneath the basement membrane, and especially round the lobules of the conglobated mucous glands, and high graded softening of the sub-mucous connective tissue form a characteristic group of anatomical changes of this disease. Erosions are far more frequent in chronic than in acute laryngeal catarrh. They present striped depressions, surrounded by very much swollen epithelium, and have a flattened, intensely red basis.

These erosions readily heal by regeneration of the epithelium, but leave behind them shallow depressions. Raginsky (*in Deutsch. Mediz., Wochenblatt, No.* 28, 1876) described a case of *ozæna* of the larynx, which was characterized by its long continuance and very abundant exfoliation of the epithelium; scabs formed, and beneath these were deep erosions. The catarrh was brought on by mycotic agencies. It belonged, very likely, to that class of laryngeal affections described by Klebs, as attributable to microsporon oris. Stork describes a form of laryngitis, which he calls Laryngeal *Blennorrhœa*, existing endemically in certain localities, and marked by its spread upon the whole mucous surface of the respiratory tract. The membrane either secretes enormous quantities of purulent and other secretions, or almost completely dries up. It assumes a long protracted and highly destructive character, but has nothing in common with syphilitic or tuberculous affections of the larynx. *Schmithuisen*, who also describes it, attributes it to mycotic influences, for he found a variety of microscopic fungi both in the secretions and in the sputa of individuals affected with it. (*Berlin. Klin. Wochenschr.*, 1878, *No.* 11.)

Loss of voice always results from chronic laryngeal catarrh. It may only amount to a slight hoarseness or to inability to speak above a whisper. Annoying inclination to cough, especially when the epithelial tissue is abraded, is likewise always present. Anatomical alterations resulting therefrom are: Dilatation of the vessels (*Duchek.*), sclerosis of the mucous membranes, more especially affecting the papillæ, as on the true vocal cords and the inferior surface of the epiglottis; and hyperplastic formations of epithelium, of different sizes and extent, upon the false vocal cords, giving the tissue a peculiar whitish granular appearance. They often penetrate into the sub-mucous tissue, and render it hypertrophic (*chorditis tuberosa*). Such hyperplastic formations are sometimes so large as to protrude into the vocal orifice in the shape of large tumors, closing up the already enlarged vocal cords, and make tracheotomy necessary. *Bruns*, *Gerhard*, *Ruhle*, *Rokitansky*, *Gibb*, and *Turk* have all described the several forms of this laryngeal hypertrophy. Granular laryngitis, like granular pharyngitis, is a result of chronic catarrh. It is both idiopathic and associated with tuberculosis and syphilis. Anatomically it is marked by the presence of distinct granulations and white transparent mucous deposit upon the posterior surface of the epiglottis, false vocal cords, and the Folds of Morgagni. Occasionally this constitutes the only symptom of chronic catarrh of the air passages.

Laryngeal Croup.

Croup and diphtheria are still considered by many to be the same disease in different forms. Klebs has already assigned to diphtheria its place among the general infectious diseases (*Arch. Exper. Patholog., Vol. IV*). As regards croup, Virchow has strictly defined its morbid anatomical character, by characterizing it as *a fibrinous exudation upon any free surface of a mucous membrane by inflammation.* It is true that there is formed occasionally a deeper-seated exudation in croupous inflammations, beside the superficial; also that in true diphtheria of the larynx a fibrinous exudate is sometimes formed upon the free mucous surface. The presence of *microsporon* diphtheriticum will always indicate diphtheria, its *absence* true croupous laryngitis. Croup is sometimes primary, and confined to the larynx, portions of the trachea and primary bronchi, or the pharynx. Sometimes it is a secondary disease, and associated with inflammatory processes in organs having apparently no connection with the larynx directly. In many cases of infectious diseases laryngeal croup forms one of the complications and a prominent symptom, and is brought about by *metastasis.*

In the primary form the whole laryngeal mucous membrane becomes covered with a yellowish or yellowish-gray membrane of about one or two lines of thickness; the anterior surface, the lateral edges of the epiglottis, and the false vocal cords are thus similarly lined. Usually a great portion of the mucous membrane of the pharynx, trachea, and primary bronchi are also covered. The pseudo-membrane is of a solid consistence, elastic; and when it is removed leaves the underlying surface very much injected and denuded of its epithelium. A new pseudo-membrane is often formed after removal of the first. Sometimes the membrane attains enormous thickness and fills up the whole laryngeal and glottal cavities. The secondary is more rare than the primary, and the exudate is usually not in the shape of a coherent membrane, but is formed in spots on the internal surface of the ary-epiglottal ligament, the sinus pyroformis, and now and then upon the true vocal cord, as soft spongy lumps of yellow color, giving to the surface a villous appearance. (*Sec. III, Tab. XII, Figs.* 1, 2.) They usually exist in connection with certain exudative processes going on in other parts of the respiratory organs (*fibrinous pneumonia*)—the heart, the kidneys, in syphilitic and tubercular affections. The metastatic form of laryngeal croup occurring in infectious diseases is generally complicated with pharyngeal croup. In scarlet fever, small-pox, morbilli, typhus, and cholera-typhoid the exudate is yellow, dry, and very brittle, easily removed from their bed. They form patches of fibrinous substances, interspersed with mortified superficial plaques, both being but slightly adherent to the mucous surface. The histological composition of the croupous exudate is identical in all forms. It consists of fibroid masses and cellular elements,

OSTEO-CHONDROMA IN LARYNX. **Sec. III** *Tab. XIV.*

Fig. 1

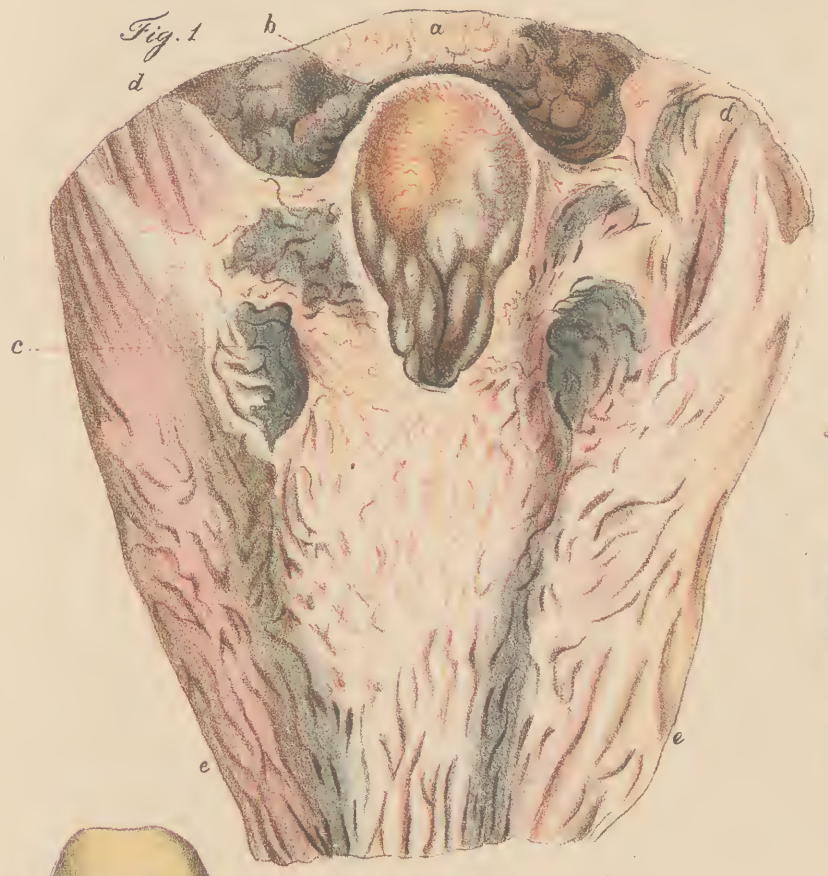


Fig. 2.

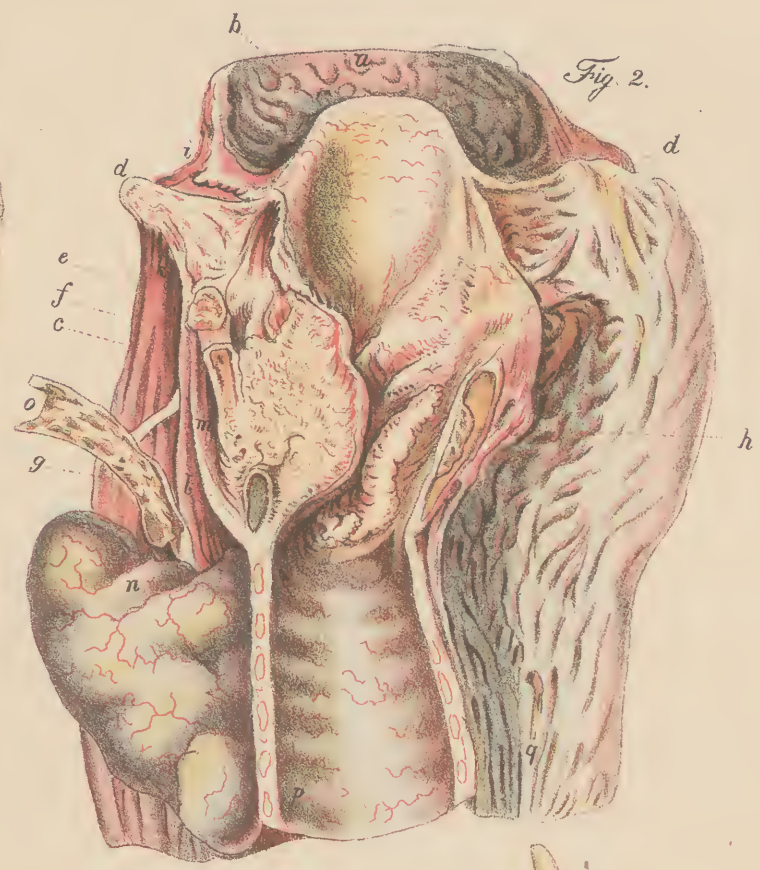


Fig. 5

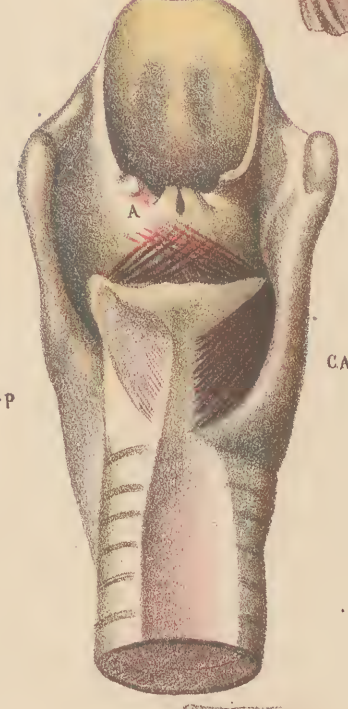


Fig. 6.

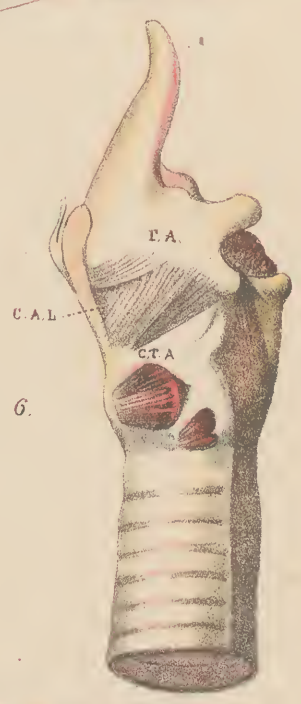


Fig. 3

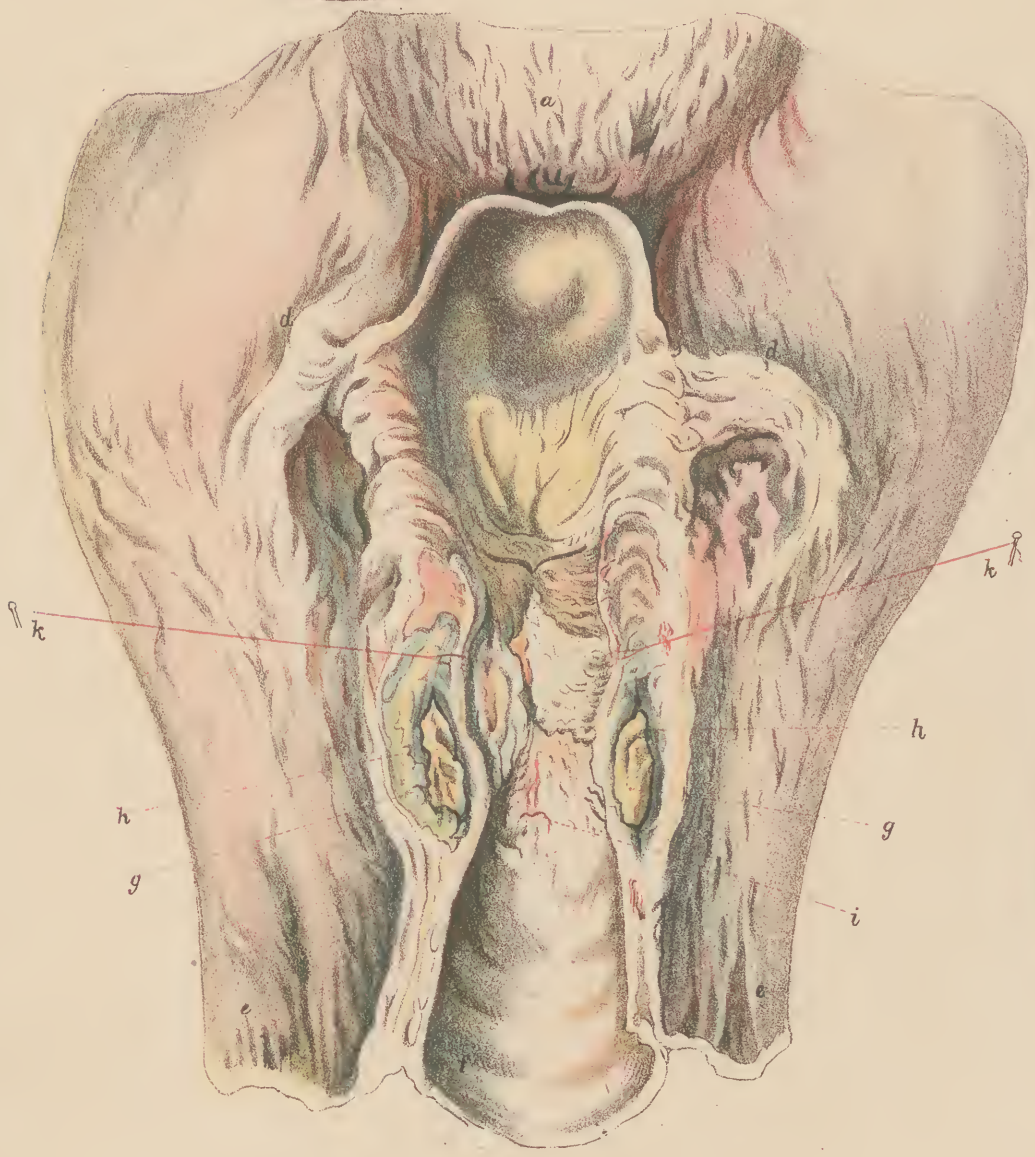
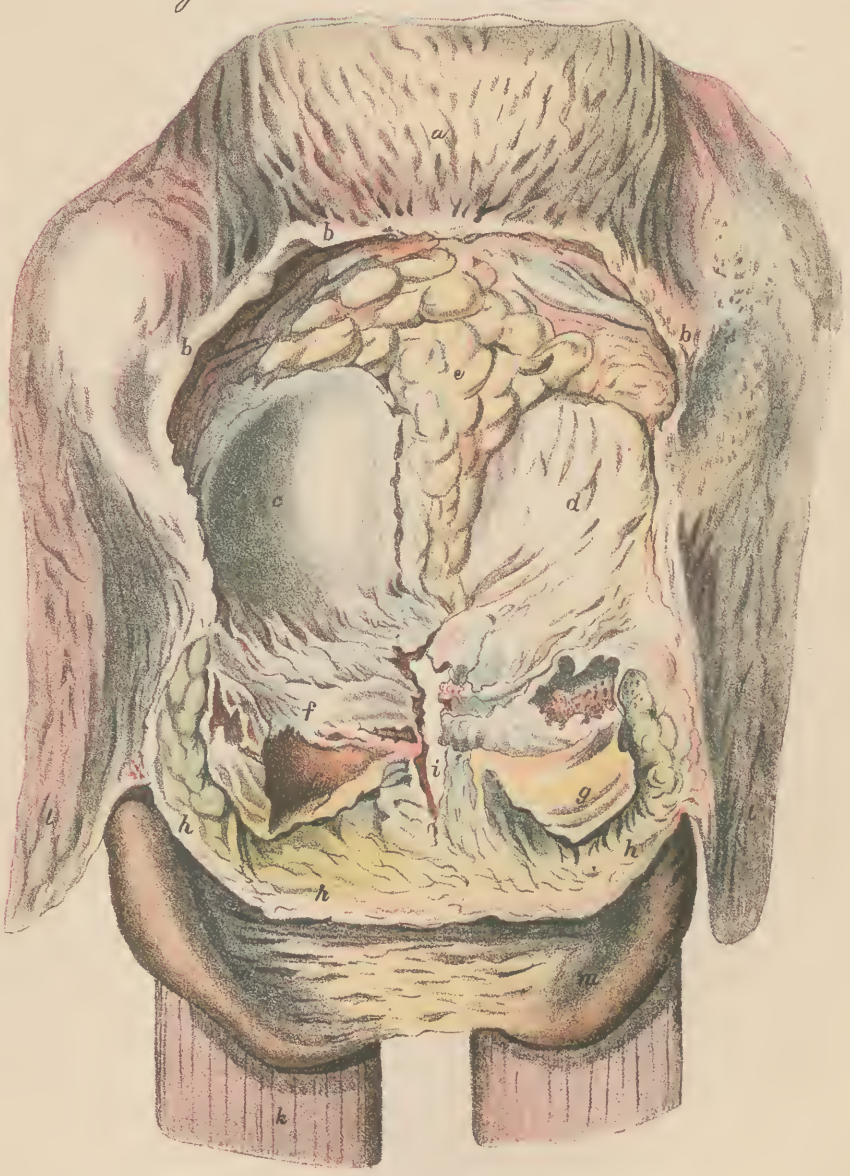


Fig. 4



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Sec. III *Tab. XV.*

ULCERATION IN LARYNX.

SYPHILITIC LARYNGITIS.

Fig. 1

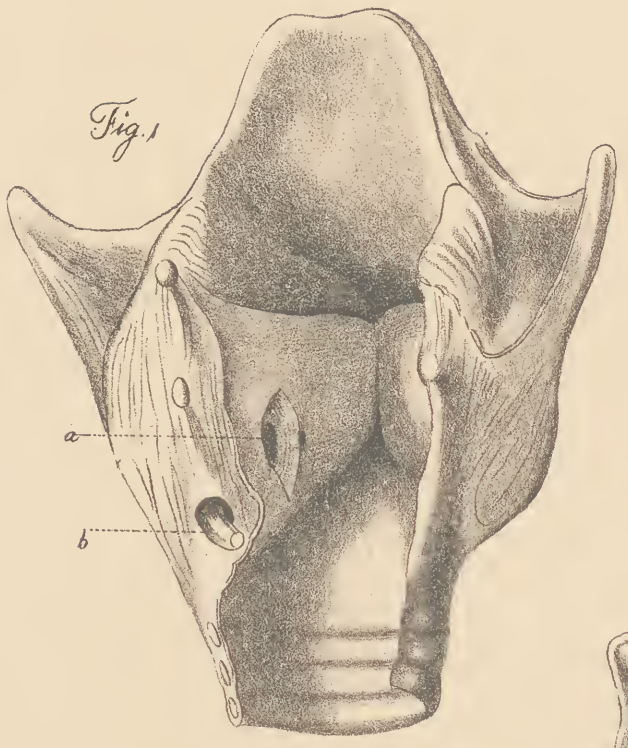


Fig. 2

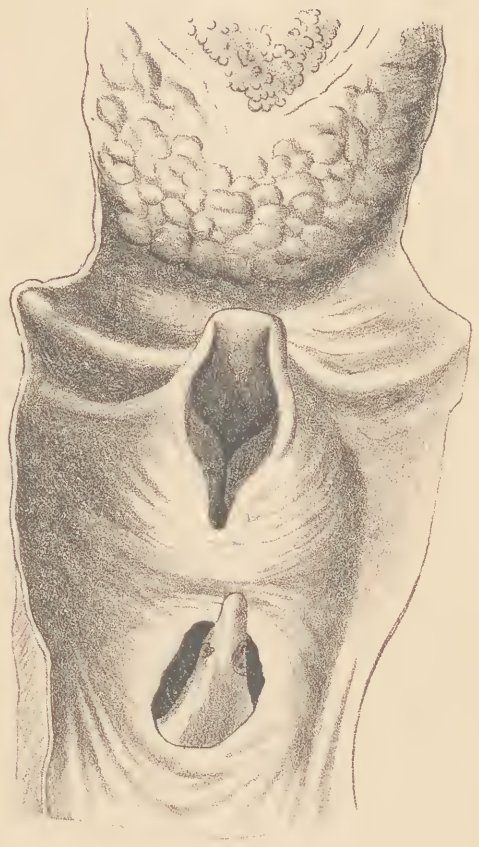
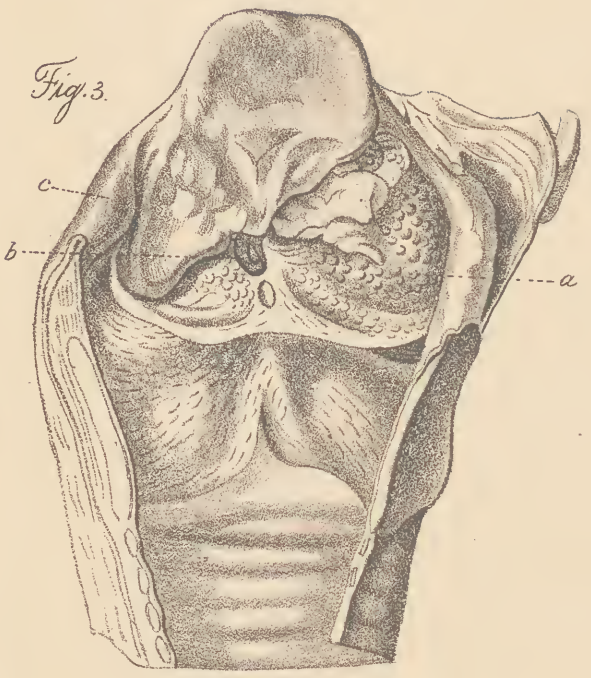


Fig. 3.



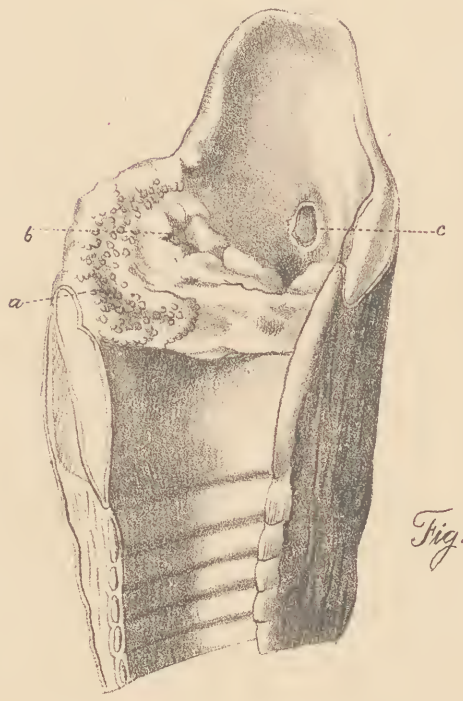


Fig. 4.

Fig. 5

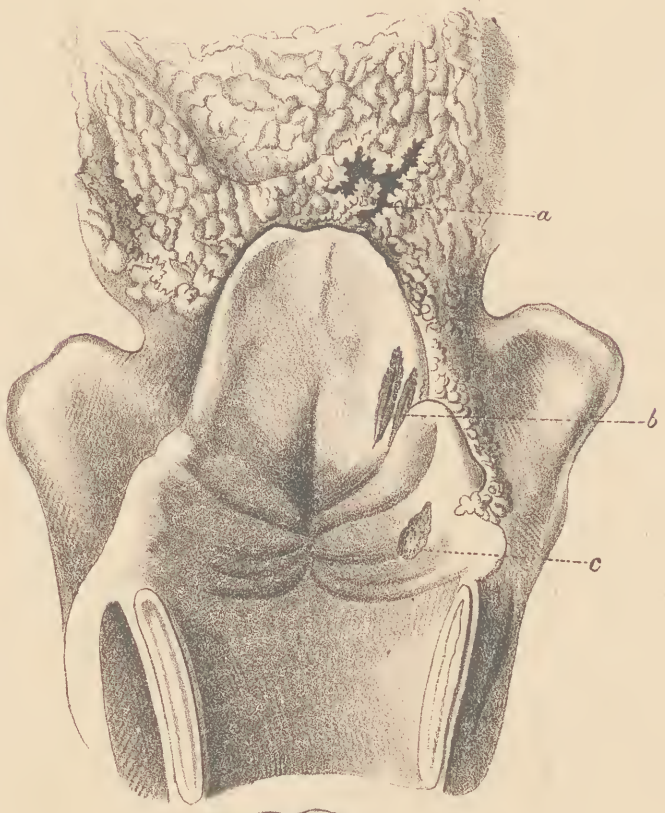


Fig. 6

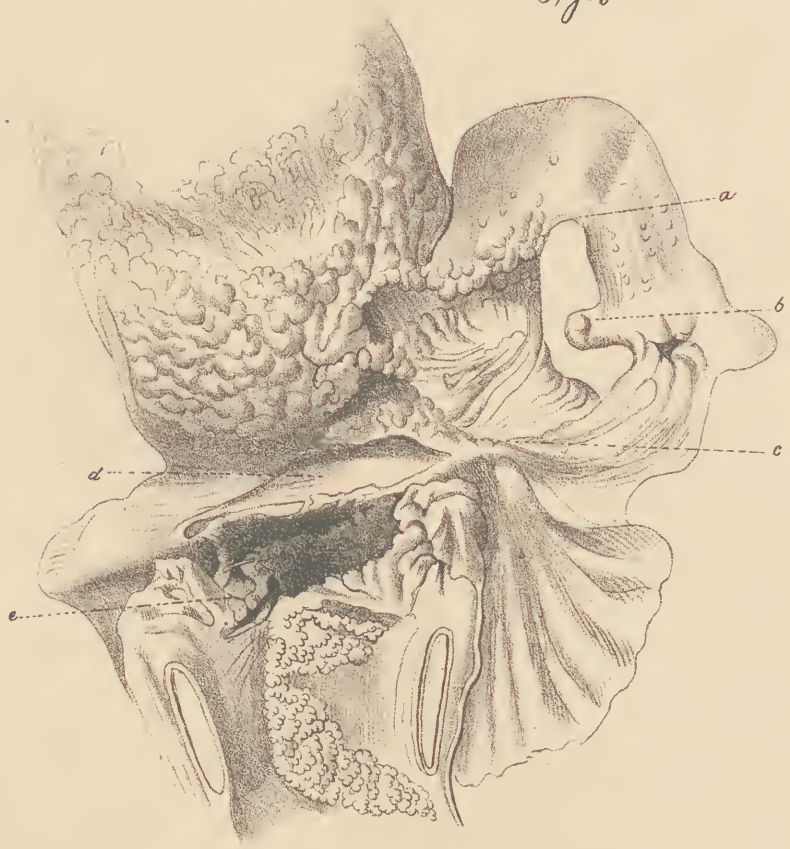


Fig. 7.

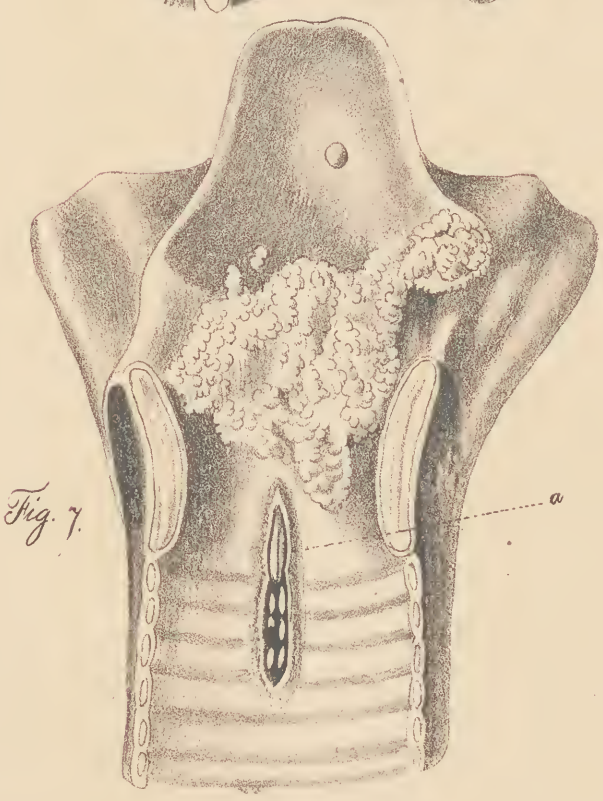


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TABLE XV.

FIG. 1.—*Perichondritis of Cricoid Cartilage.* Beneath the vocal cords a thick layer of connective tissue is formed, visible at (a), the cartilage is obliterated and only a portion of it is left attached at (b). The connective tissue around it is thickened. It formed a stricture of the air passages, leading to death by asphyxia.

Fig. 2.—*Posterior aspect of Larynx.* The thyroid and cricoid cartilages are destroyed by perichondritis and abscesses consecutive to typhoid laryngitis. The laryngeal orifice is constricted by considerable infiltration into the ary-epiglottal folds. The exudate was emptied into the œsophagus, which it had perforated, and through the orifice thus formed a portion of the cricoid cartilage projects.

Fig. 3.—*Extensive laryngeal ulceration associated with pulmonary tuberculosis.* The ulcers have destroyed the right ary-epiglottal fold, the right vocal cord, the right half of the epiglottis and the right Fold of Morgagni, and a great portion of the left vocal cord. On the bottom of the ulcers connective tissue nodules are visible at (a). At (b) a densely swollen part of the left side of the orifice of the larynx projecting upon the apex of the arytenoid cartilage (c).

Fig. 4.—Tubercular ulcer over the region of the left arytenoid cartilage and very numerous excrescences at (a). At (b) abrasion. A smooth base and projecting rims of the ulcer, beginning to cicatrize. A circular ulcer on epiglottis (c).

Fig. 5.—*Larynx of a man affected with syphilis and pulmonary tuberculosis.* In the pharynx and on the uvula cicatrices and recent ulcers. On the base of the ulcers with hyperæmic basis and sharp edges of irregular form at (a). The mucous membrane covering the arytenoid cartilage is swollen, the connective tissue infiltrated. On the lower surface of the epiglottis there are two ulcers (b). On the surface over the vocal cord another abscess (c). The death of the individual was caused by hæmoptysis.

Fig. 6.—*Very extensive destruction of larynx by syphilis.* (a) Velum palati drawn to one side, the uvula is destroyed. (b) Cicatrix of destroyed portion of the pharynx. (c) Cicatricial contraction of the mucous surfaces converging at (d). (e) presents the remnant of the destroyed epiglottis.

Fig. 7.—*Villous cancer in the larynx.* An opaque white mass constituting very numerous vegetations upon the mucous surface, nearly closing the glottal opening. At (a) new formation of vegetations on the free surface of larynx. Tracheotomy was performed. The individual died of suffocation.

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the relative quantity of each and their relation to the mucous membrane differ under different circumstances. The fibroid portions form a network of different sized fibres, and in their meshes, which also vary in dimension, there are found exudate cells. The number of cells is usually in inverse ratio to the quantity of the fibroid substance. In primary croup the threads are thicker, and a lesser number of cells exist; in secondary the threads are very fine, and a greater number of cells are contained in the meshes. In the metastatic form both exudated cells and decayed epithelial cells fill the meshes. Both fibroid masses and exudate cells are derived from the blood, as *Weigert* has experimentally proven. The deeper layers of the mucous membrane are always more or less infiltrated with cellular elements, which occasionally fill up the sub-mucous intermuscular spaces. The mucous glands are filled with secretion and their epithelial lining granular. The causes of croup may be mechanical, chemical, or living *irritants*, which are capable of producing that peculiar form of fibroid exudation upon the surface of a mucous membrane, and at the same time cellular infiltration into its deeper layers. Secondary and metastatic croup are attributed to *microsporon septicum* by *Klebs*. *Fischel* states that he discovered great numbers of monadinæ in croupous membranes. *Bohn*, *Gerhard*, and *Schweninger* have shown it to be of infectious origin. The termination of the disease differs in diopathic and secondary. In the first, the formation of extensive membranes in the air passages, which usually happens in little children, is usually fatal (although occasionally saved by laryngotomy.) The narrowness of the glottis, the want of power, the excessive prostration which so speedily supervenes, the quick spread of the exudate upon the trachea and bronchi hinder the removal of the exudate from the air passages, and bring on speedy asphyxia. In the secondary and metastatic form, the membrane being neither continuous nor so adherent, nor is it so speedily formed, danger from suffocation seldom exists; and when the patient makes any efforts the exudated masses are readily removed. The infiltration of the sub-mucous tissue, and the decomposed and gangrenous nature of the exudate and of the tissues, alone form dangerous contingencies. Cessation of the irritated state of the sub-epithelial layer brings about loosening of the croupous masses, which are easily removed by coughing or otherwise. There is usually complete restitution of the epithelial covering, unless the tissue has undergone very extensive destruction, and the deeper layers have become quite altered by excessive infiltration. The regeneration of the epithelium begins in the nearest unaffected part of this tissue, which gradually spreads over the denuded ones in the same manner as the epidermis is restituted over granulating cutaneous tissue from adjacent unaffected portions of the epidermis. (*Klebs and Arnold.*)

Laryngeal Diphtheria.

That diphtheria is produced by micro-organisms is a well-established fact. It usually affects children of both sexes far oftener than adults, and of these those who attend and nurse children are far more liable to be affected with it than others. Whether the diphtheritic *microsporon*, as it has been named by Klebs, is peculiar to this alone or not, is not yet quite settled. That it has been found in the blood, in the exudates, in the kidneys of persons affected with diphtheria would seem to indicate its specific nature. It possesses the power to destroy the mucous membrane, and is the cause of the exudation through which it extends locally and diffuses through the whole system. For not only does it increase in the destroyed mucous membrane, but penetrates into the whole circulation, from which it is eventually driven out by the kidneys, but not before it has produced more or less morbid alterations and great functional disturbance in those organs. The microscopic appearance is shortly described by *Thierfelder* (*in his Atlas of Patholog. Histology*) as follows:

"In the many-layered pavement epithelium there is lifted up like a flat ridge—interrupting the continuity of the upper stratum—a number of exceedingly fine granular layers, irregularly placed one upon the other, the lowest stratum sending a number of projections into the sub-epithelial structures. Beneath these, and surrounding them, are vessels highly dilated; near them are some few emigrated colorless corpuscles. The connective tissue fibres are pressed asunder by sero-purulent exudations, and appear soft and anæmic. The acini of the mucous glands are equally infiltrated with serum." The croupous patches very often combining with it present the following appearance: "The basement membrane imperceptibly passes into a thin purulent layer; facing the epithelium is a strongly marked line. In place of the epithelial tissue, which has undergone fibroid alteration, and has been obliterated, there is a network of very fine meshes formed by highly refractive and very thin fibres. Within the meshes are found a few well defined epithelial nuclei, a great many granular degenerated colorless blood corpuscles, and finely grained detritus." In consequence of the removal of the epithelium in whole layers, the microsporon penetrates into the mucous tissue proper, and often forms, very speedily, a coherent and very massive exudation. The time it takes from the initial state to complete formation of the extensive exudation is from three to twenty hours. After this it gains constantly in thickness. (*Letzerich die Diphtherie.*) Diphtheritic patches in other cavities differ from these in their containing more pus corpuscles. In the larynx the patches are most frequently only found upon the upper sections of the mucous membrane situated above the vocal cords. They are softer than in the upper portions. The fungi are capable of penetrating not only into abdominal viscera, and then produce characteristic diphtheritic patches (Lange), but also into the lymph vessels and lymph glands, and thus spread over the whole organism. In the kidneys they produce well characterized changes. Croupous exudations, as often as they appear, are only in laryngeal diphtheria; in pharyngeal, nasal, buccal, glossal diphtheria they are never produced. (*Letzerich.*)

Symptoms and Course of the Disease.

Besides redness and swelling of the tonsils in the early stages, there is nothing remarkable in the local condition. When the swelling is considerable it produces a very transitory feverless angina. When the surface of the tonsils becomes white and opaque, there is an intermittent sense of pain in the throat, increased in swallowing. The pain often extends into the internal and middle ear. Difficulty of and pain in swallowing increase steadily with the formation of the diphtheritic patches and concomitant exudation. Upon the much-swollen tonsils appear large projecting plaques, which are often removed by strong cough or strangulation in the attempt to swallow anything. They are of a very dirty yellow or grayish color, and often produce vomiting when they happen to lodge in the fauces. The patient's breath is extremely fetid. Very often swelling of the sub-maxillary glands, extending to the surrounding soft tissues, causes a great deal of pain. The swollen tonsils are usually also very painful. Rigors and alternate sensations of heat and chilliness exist at first. Should formation of new patches or reformation of those removed take place, it will indicate a general infection of the body, and the symptoms will often change very suddenly to a very high grade of fever, often delirium, flushed face alternating with extreme paleness, injected conjunctiva, headache, vomiting occasionally intestinal tenesmus. Children, otherwise very lively, lie very quietly or in a stupor, and indifferent to everything going on around them. In most cases constipation and meteorisme exist. The seat of pain is usually around the navel. Partial suppression, or very scanty urination, and pain on pressure upon the region of the kidneys. The urine contains more or less albumen. The microscope shows fibroid casts and vast numbers of microsporon in different groups and several stages of development. Convulsions very often attack young children—they are sometimes the first symptoms alarming parents and nurses, when others have been overlooked; at first they are intermittent, but gradually become continuous, until coma and general anæsthesia set in. As a rule diphtheritic nephritis sets in after development of diphtheritic patches, and croupous exudations have formed upon the respiratory surfaces and other mucous membranes; or these may form subsequent to the nephritis. When massive laryngeal exudations become complicated with the diphtheritic process death is usually the result, and that very speedily. Paralysis of the vocal cords is often associated with diphtheria and may persist for a long while after recovery from the first. Motor paralysis of the lower extremities and occasionally of the upper are often the result of the general toxicæmia of this insidious disease. (*Letzerich.*)

Œdema Glottidis, Sub-mucous Laryngitis of Cruveilhier.

It is characterized by an abundant accumulation of liquid transudate in the sub-mucous connective tissue, the fibrous bundles of which become much separated from each other and also filled with serum. There is a chronic and an acute form of this disease. The last occurs in the vicinity of acutely-inflamed tissue, is local, and generally assymetric. The mucous membrane in the œdematous part is turgid and pale, and is surrounded by tissue in a well marked state of inflammation. It is of solid consistence, and only discharges its liquid contents on pressure after cutting through it. The transudate is made up of a slightly turbid coagulum, in which a number of lymphoid bodies are suspended. Chronic œdema usually affects both sides, especially the ary-epiglottal ligaments,

TABLE XVI.

LARYNGOSCOPIC VIEWS.

Fig. 1.—Mucous polypus on the free edge of the right true vocal cord. Fig. 2.—Fibroid polypus on the left vocal cord, its upper portion œdematous. Fig. 3.—On the right true vocal cord a conical dense fibroid polypus, on its base a circular ulcer. Fig. 4.—A large polypus filling the upper laryngeal space and the Folds of Morgagni. Fig. 5.—A fibroid below the vocal cords attached to the anterior laryngeal wall. Softening and pendulous condition of the vocal cords. Fig. 6.—Nearly the whole posterior surface of the larynx is covered with pappiloma forming two layers, and in a state of active vegetation. Fig. 7.—Ulceration along the edge of the left vocal cord, dividing the fibrous covering of the cord. An ulcer existing between the arytenoid cartilages indicates tuberculosis of the larynx. Figs. 8 and 9.—Ulceration of the whole inner surface of the false vocal cords. Fig. 8, early stage, Fig. 9, later stage of the disease—both in tubercular condition. Fig. 10.—Nodular infiltration of the true vocal cords. A deep ulcer on the posterior aspect of the arytenoid space. Fig. 11.—Tuberculous destruction of both vocal cords. Fig. 12.—Extensive tuberculous destruction of all internal parts of the larynx caused by sub-acute tuberculosis. Fig. 13.—A fissure in the internal posterior wall of the otherwise healthy larynx. Fig. 14.—Swelling on the internal posterior wall of the larynx an early sign of tuberculosis. Fig. 15.—A catarrhal ulcer on the right vocal process. Its wall is raised by epithelial cell division. Usually a sign of pulmonary phthisis. Fig. 16.—Catarrhal ulcer on the left vocal cord in acute laryngitis. Fig. 17.—Tuberculous destructive ulcers on both vocal cords in rapidly progressing tuberculosis. Fig. 18.—Œdematous swelling of the epiglottis, the left ary-epiglottal ligament, and the left arytenoid cartilage; a case of slowly developing tubercular infiltration. Fig. 19.—A view into the whole length of the trachea and the bifurcation of the bronchi (healthy). Fig. 20.—An enormous polypus passing from the right vocal cord posteriorly, nearly filling the whole cavity of the larynx. Fig. 21.—Stricture of the larynx by a pseudo-membrane below the vocal cords. Fig. 22.—Cicatrices after syphilitic ulceration; the epiglottis partly destroyed, in the right false vocal cord a deep hole, adhesion to the true vocal cords. Fig. 23.—Total destruction of the larynx by syphilis. (Speedy death.) Fig. 24.—Epithelial cancer on the right vocal cord.

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the anterior surface of the epiglottis, and in rare cases the true vocal cords. (*Sec. III, Tab. XII, Figs.* 4, 5, *acute epiglottitis.*) Very rarely is the entire mucous surface affected. The portions thus involved are formed into thick ridges or rolls, very tense, of a yellow transparent jelly-like appearance, and have a glistening surface. When the epiglottis becomes œdematous it rolls up into a swollen cylinder (*Fig.* 5, *same Plate*), and becomes very visible in the pharynx and is felt by the patient like a large lump, which closes up the pharyngeal and laryngeal cavities. The most dangerous is dropsy of the ary-epiglottal ligaments, for not only does it cause a closure of the upper portion of the larynx by swelling more than any other part of the mucous membrane, but also by its being drawn into the glottal orifice during inspiration. The least dangerous is œdema of the Folds of Morgagni. The infiltrate in the chronic state is very clear, and contains very numerous albuminous and fat granules and a few lymphoid bodies. The mucous glands are dilated and filled with the same transudate, the epithelium is exfoliated in a great measure. In the acute form the mucous glands are compressed. In the acute form the mucous membrane is tense; in the chronic form the sub-epithelial layer is filled with lymphoid cells and degenerated epithelium. Croup, laryngeal diphtheria, perichondritis, acute laryngeal catarrh, phlegmon of the larynx with diffuse pharyngitis, of the parotid glands, and many other diffuse inflammations of the cervical tissues are the causes of acute œdema, which often causes very speedy death. High-graded œdema forms part of general dropsy. It is less dangerous than the acute form. In rare cases the dropsical liquid becomes absorbed, and the collapsed membrane is then thrown into numerous folds. A few histological remnants are left behind.

Phlegmonous Laryngitis.

This very much resembles acute laryngeal dropsy, but differs from it in the following: That there is here a tough, mostly purulent, infiltration into the sub-mucous tissue. Only portions of the larynx become thus affected. But the swelling is never so massive, the parts become tough, plump, slightly discolored; the transudate forms a coagulum, and is mixed with pus. There are formed submucous abscesses, which raise the mucous tissue above them, and eventually break through it, forming deep, undermining ulcers. The inflammation will then often extend deep into the perichondrium, calling forth quickly extending perichondritis. (Sec. III, Tables XIII, XIV, *many of its figures.*) Serous infiltration is very often formed in the soft adjacent tissues. Both conditions produce dangerous constrictions of the respiratory passage. Enormous quantities of exudated cells are found in the inter-fibrous spaces of the sub-mucous connective tissue. Slight quantities of infiltration are usually very readily absorbed; great quantities, containing much pus, usually produce perforation by suppuration, and the matter will penetrate into the laryngeal, pharyngeal, and œsophageal cavities, or empty outside through the skin. (Sec. III, Tables XIII, XIV, etc.) By septic agencies the destruction becomes enormously extensive, and usually ends fatally. Under favorable circumstances there are formed cicatrices in the tissues, and partial restitution of the organ finds place. (*Henle and Pfeufer. Zeitschrift f. Ration. Medezin., Series III, Vol. I, p.* 237.) This lesion is generally secondary to other inflammatory and infectious primary diseases.

Perichondritis.

This disease, which has several synonyms, is, strictly speaking, an inflammation of the perichondrium, resulting in suppuration and formation of abscesses. The perichondrium of the larynx, owing to many circumstances, is more subject to inflammations than many other similar structures. Even normally, in old age, this tissue undergoes a process of vascularization and ossification (Eppinger). Only portions of this tissue are attacked by inflammation. *Tobold* says that the thyroid, cricoid, and arytenoid cartilages are frequently affected by perichondritis. Friedreich claims the greatest frequency of this inflammation to exist in the arytenoids, whilst Tuerk maintains that the cricoid cartilage is as often affected as the others. All cartilages are never affected at once. There is an idiopathic and a consecutive or secondary perichondritis. The first is of most rare occurrence. In all there is formed a puriform infiltration into the perichondrium, which thereby swells up. The infiltrate detaches it from the cartilage and fills up the space between them. (Sec. III, Tab. XIV, Figs. 3, 4.) Tumors formed thereby project either outward toward the surrounding tissues, or into the laryngeal cavity. (Sec. III, Tab. XIV, Figs. 1, 2.) Very soon the exudate is changed into pus and constitutes an abscess, which still further separates the membrane from the cartilage and the space is still more filled up with pus. (Sec. III, Tab. XII, Figs. 3, 4.) Evacuations of such abscesses differ according as the soft parts are pierced by the corrosive pus, or the cartilage is perforated by necrosis from lack of nutrition. In the first case the liquid pus only is discharged, the fundus of the abscess becomes a corrosive ulcer, which eventually affects the cartilage, and causes infiltration into the surrounding tissues. In the second case the spread of destruction of the cartilage will continue for some time, until portions of it become obliterated. Necrosed parts of a cartilage in such a condition will either be loosened from their surroundings and detached by exfoliation, or their obliteration be slowly brought about by suppurative caries. In very rare cases, healing of such wounds is effected by suppuration, granulation, and subsequent cicatrization. *Schottelius* has lately observed a case of spontaneous cure of a case by production of a funnel-shaped cicatrix upon the surface of the cartilage.

Alterations of the laryngeal tissues in typhoid fever differ in degree and extent according to the intensity of the morbid process of the typhoid infection. There may exist very superficial affections of the laryngeal mucous membrane directly connected with general derangements in that tissue throughout the body from the typhoid miasm, or in connection with lobular pneumonia, a most frequent complication in that disease. There may be acute catarrh, hæmorrhages, superficial erosions or surface mortification. With the last lesion very extensive and very deep ulcerations are often connected, and are marked by a mycotic character. There may be infiltrations similar to those forming in the intestines, such as nodular or granular, and ending in gangrene and ulceration. Fibrinous exudation of a croupous kind often takes place as a consecutive lesion. The catarrhal process in the laryngeal mucous membrane, although less severe than in ordinary catarrhal processes, leads, nevertheless, to denudations of the many portions of the cartilages, by severe exfoliation and gangrene of the epithelium. The edge of such a denuded cartilage projects between two shrunk mucous surfaces. Typhoid diphtheritis is another form of desquamative laryngeal catarrh in which exfoliated epithelium is found in yellow, smaller or larger flakes, mixed with gangrenous detritus and enormous masses of micrococci. They are usually in the folds of the loose mucous surface in the epiglottal region. This form of mycosis often leads to gangrenous ulcers, and necrosis of the cartilage of the epiglottis, or the other upper cartilages. (*Tuerk, Seibert, Krafft, Gerhardt, Eppinger,* have all described those very numerous deep alterations produced by mycotic agencies, but which could only be lightly indicated in these few pages.

Alterations in the Larynx in Variola.

In all cases of small-pox there are more or less intense and extensive changes in the larynx. B. E. Wagner states that of 170 cases of small-pox he found true pustules in the larynx in 144. Rokitansky, Foerster, Tuerk, Schroetter, and Ziemssen positively claim to have found pustules in the larynx of persons affected with variola. Eppinger, basing his opinion on histological researches as thoroughly made by Weigert (in *Anatomische Beitraege*), Klebs, and Luginbuhl in 1873, flatly denies the possibility of the existence of true variolous pustules in the larynx. "But," says he, "it must be conceded that examination of the tissues merely with naked eye leads to *supposition* of their existence in those parts." In the early stages the mucous membrane is uniformly injected, moderately swollen, and but slightly covered with mucus. Upon the epiglottis, the arytenoid cartilages, and the true vocal cords, very small, white, discolored spots, having very sharp outlines, make their appearance. They look like nodules, yet do they barely project above the level of the mucous surface. Under the microscope they prove to be but superficial alterations of the epithelium, covered by the most external layer of that tissue. Deeper down in the tissue the cells lose more and more of their structural forms and at last form a heterogeneous lump of cellular detritus. In the more advanced stage of the disease, even the outermost layer of the epithelium becomes likewise altered, and the whole is raised above the level of the adjacent structure. The little raised lump becomes filled with lymph serum, and resembles a pustule. Klebs succeeded in finding numbers of micrococci in this little lump, and ascribes the whole alteration to their agency.

The morbid changes in scarlet fever and measles are mostly exceedingly intense hyperæmia, ecchymosis, and violent, acute catarrh of the larynx, which, according to *Tobold*, usually precede the respective eruption of the skin. Œdematous swelling often accompanies the catarrhal process and often leads to stricture of the larynx. (*Ziemssen, Stoffell.*) Gangrene and fibrinous interstitial exudations are of rarest occurrence in these diseases.

LARYNGOSCOPIC VIEWS. **Sec. III** *Tab. XVI.*

Fig. 1.

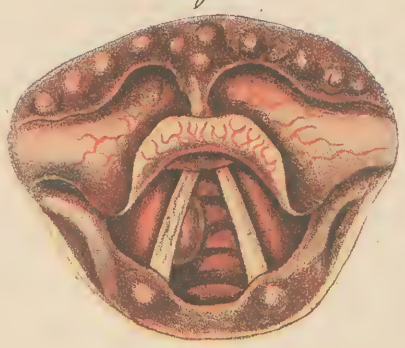


Fig. 2.

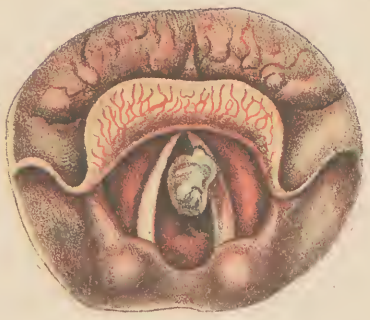
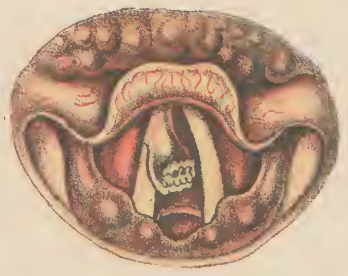


Fig. 3.



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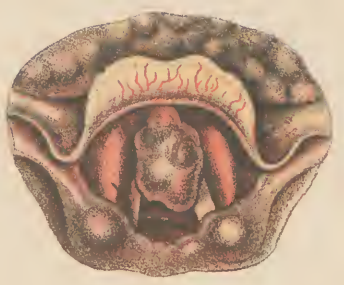

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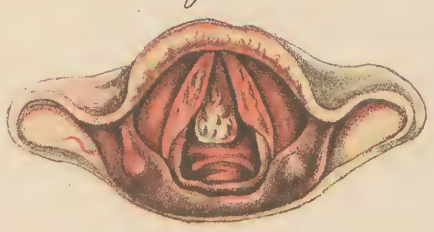

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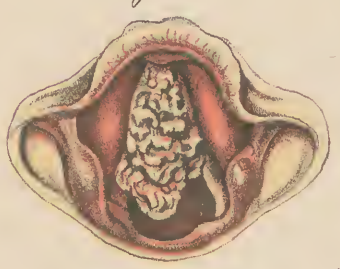

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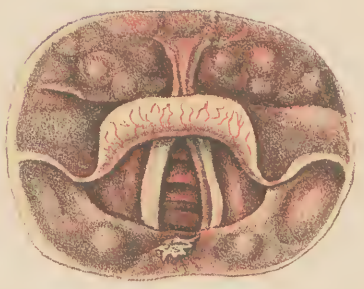
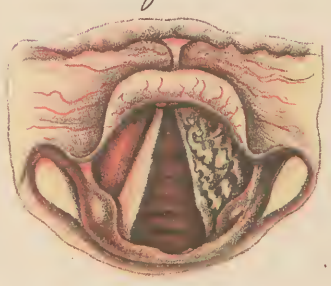


Fig. 8.

Fig. 9.



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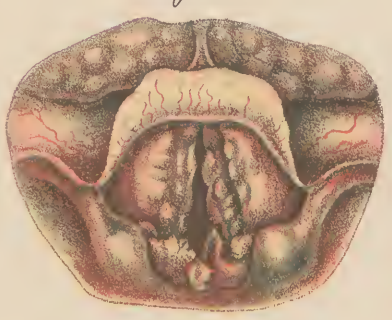

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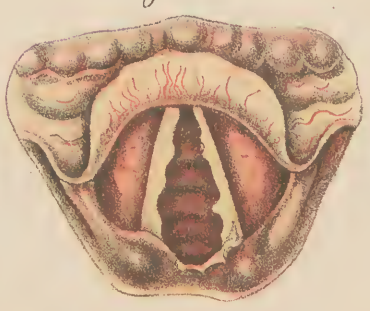

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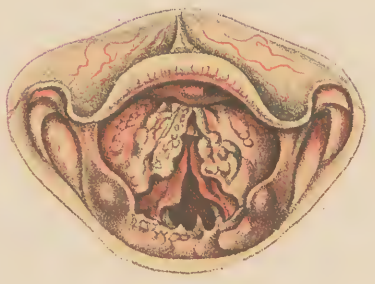

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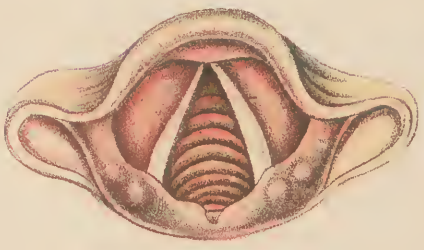

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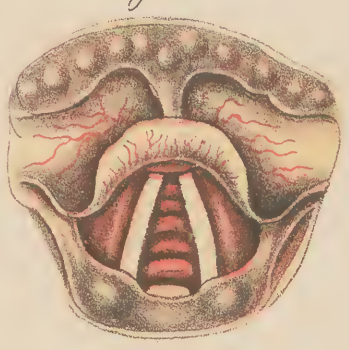


Fig. 15.

Fig. 16.

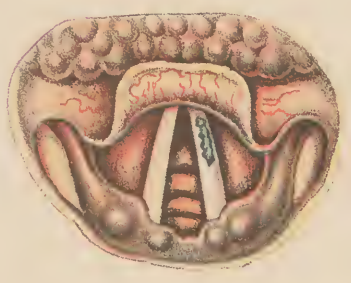
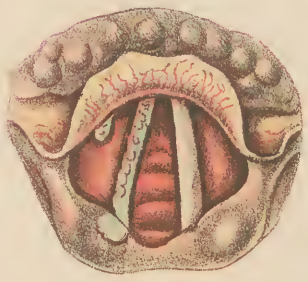


Fig. 17.



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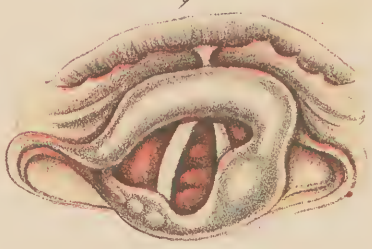


Fig. 19.

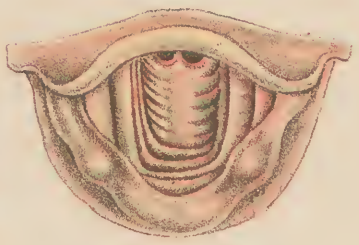
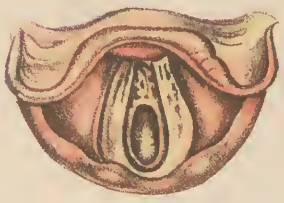


Fig. 20.

Fig. 21.



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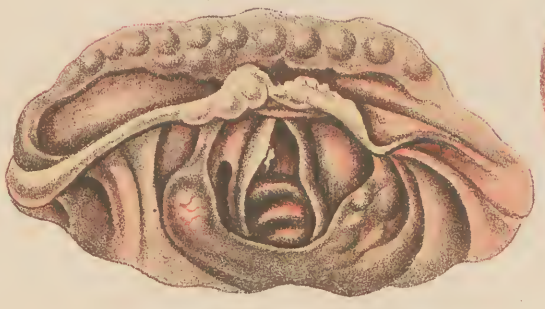


Fig. 23.

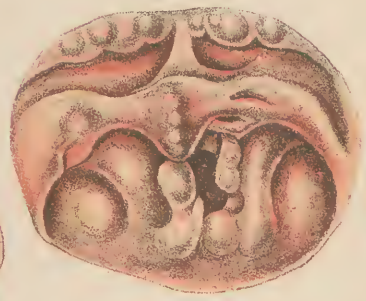
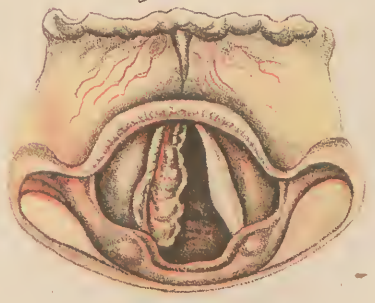


Fig. 24.



W. M. DONALDSON & CO. LITH. CIN. O.

DISEASES OF THE BLOOD VESSELS.

EXCESSIVE DILATATION OF THE LEFT VENTRICLE.

ULCERATION OF THE AORTIC SINUS.

Sec. IV. *Tab. I.*

Fig. 1.

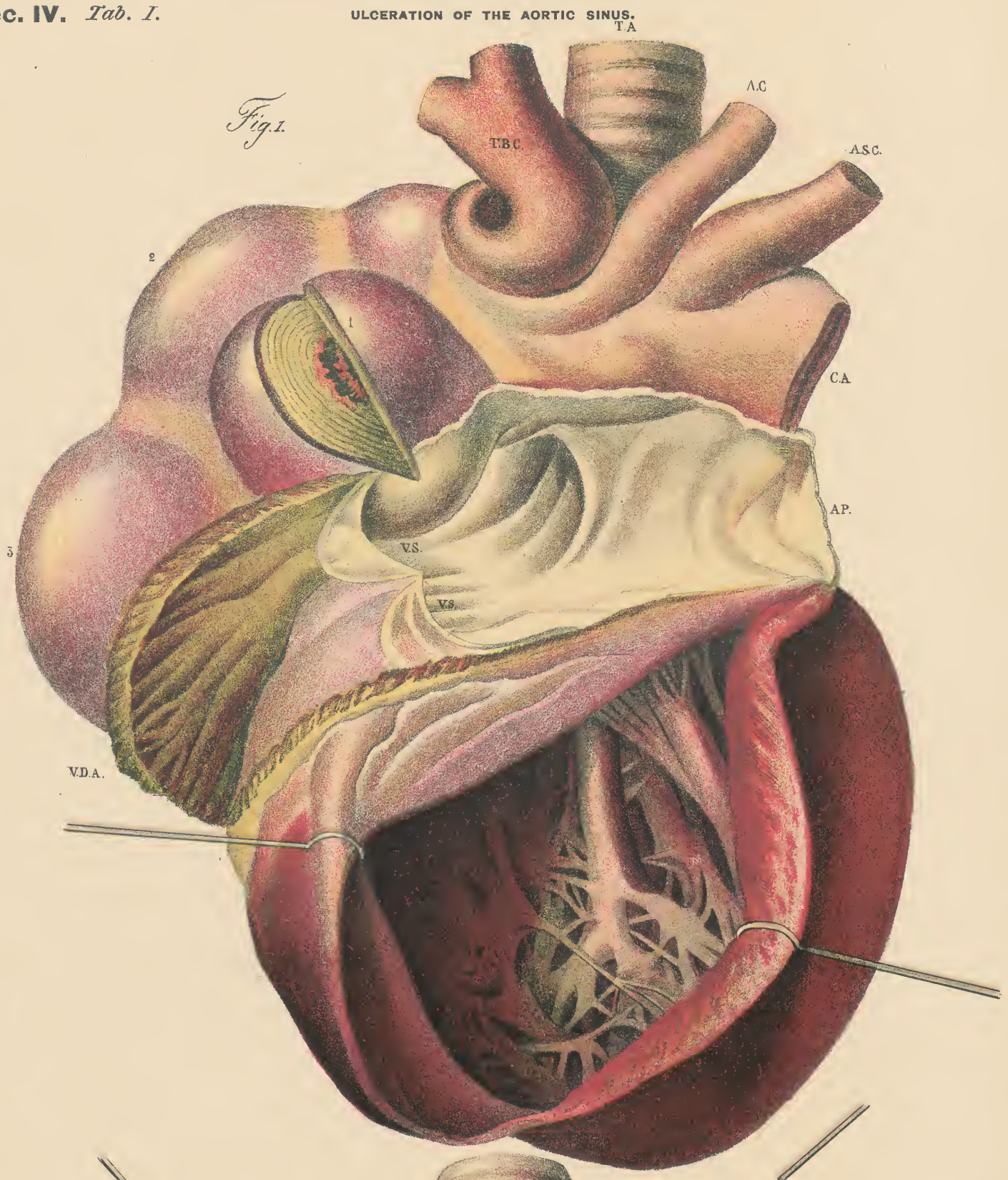
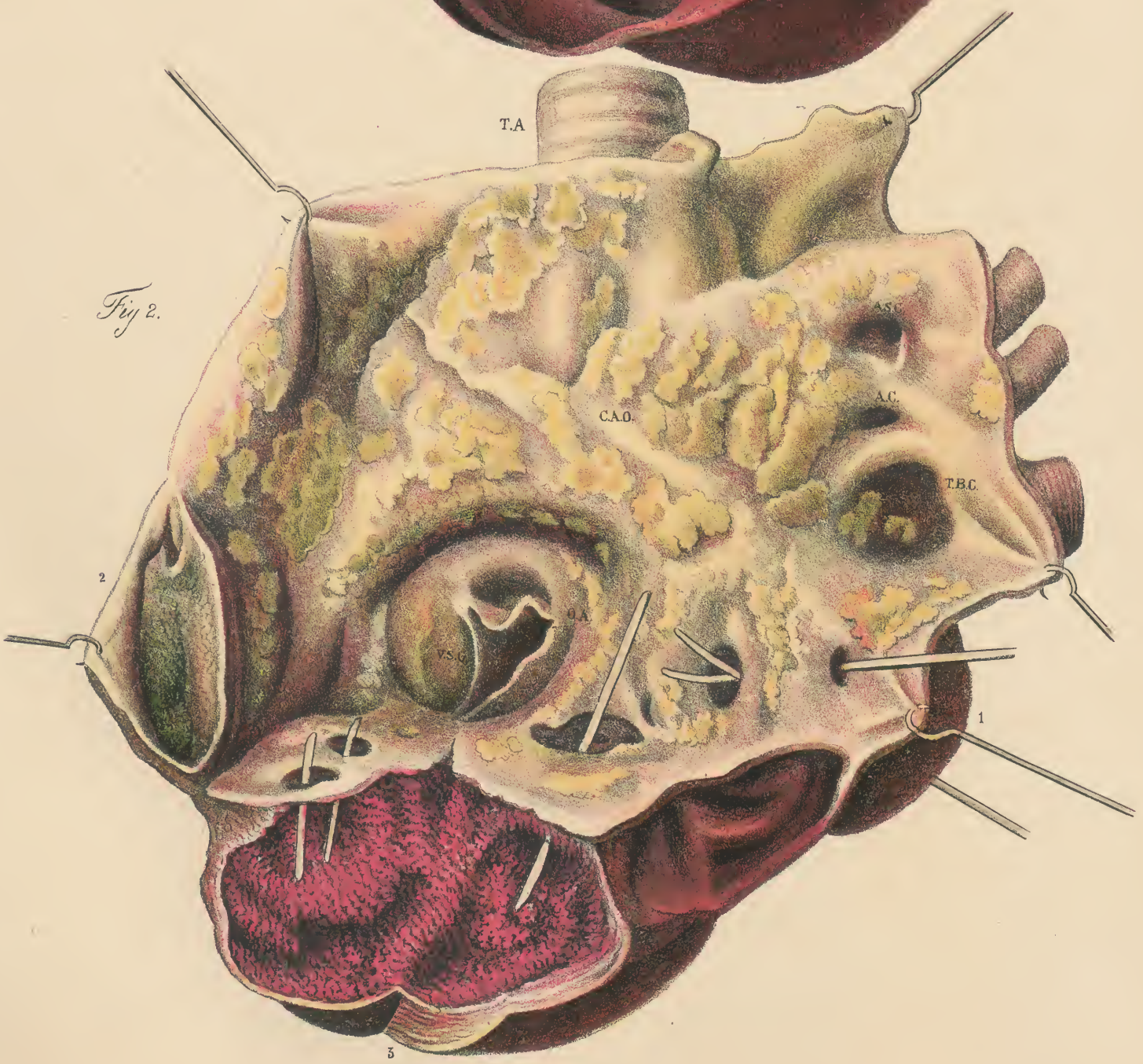


Fig 2.



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Section IV. Table I.

Hypertrophy and Dilatation of Left Ventricle. Aneurism of the Origin of the Aorta.

Figs. 1 and 2 represent the heart and the aorta of an individual who died suddenly. No previous history of the case. Fig. 1, the right ventricle (*V. A. D.*), as compared with the left, is exceedingly reduced in size, while the left is enormously hypertrophied and very much dilated. The base of the right ventricle passes forward as a funnel-shaped prolongation, and gives origin to the pulmonary artery. In the ascending portion of the arch of the aorta, beyond the origin of the innominate artery, there are three aneurismal enlargements (Nos. 1, 2, 3,). No. 1 is laid open, and presents the well-known stratified disposition of the inside of an aneurismal sac. The enlargement protrudes into the pulmonary artery from without inward, just above the semilunar valves (*V. S.*, *V. S.*), and then communicates with the interior of this vessel. Fig. 2 represents the ascending portion of the aorta opened. No. 1 shows the dilated portion of the vessel, No. 2 the yellowish-white, calcareous and sclerotic plaques on its interior surface; No. 3 represents the origin of the aorta (*O. A.*), its healthy semilunar valves (*V. S. G.*), and the aortic sinus. In Fig. 2, the aneurismal sacs 2, 3, are opened. No. 1 communicates by two orifices with the cavity of the aorta; Sac No. 2 is open but empty, No. 3 is widely opened and shows its communication with the artery by three openings, indicated by stillettes passing through them.

[Continued from Page 8, Section II.]

H. Stricture of the left auriculo-ventricular opening. The following are its physical symptoms:

1. *Presystolic murmur* over the apex, which is often palpable as a presystolic fremitus. Occasionally, instead of the murmur, a partial diastolic tone will be heard. The second aortic tone is very feeble, whilst the first ventricular tone, over the apex, is very loud.

2. *Dilatation of the right ventricle* will be produced by the stricture in the left heart, causing enormous resistance in the pulmonary circulation, as stated above.

3. *Hypertrophy of the right ventricle*, the compensatory hypertrophy will increase the force of the second pulmonary sound.

I. Stenosis of the pulmonary orifice is connected with the following changes: 1. Systolic murmur over the pulmonary artery. It is very loud and extensive, and is felt as a systolic fremitus; the second sound is low.

2. *Hypertrophy of the right ventricle*, due to circulatory impediment. As a clinical symptom *cyanosis is here very significant.*

K. Incompetency of the pulmonary valves. Physical symptoms are:

1. *Diastolic murmur* over the pulmonary artery, sometimes palpable as a fremitus. It may spread to other orifices of the heart, and even to the arteries of the neck.

2. *Dilatation of the right ventricle* by over-filling it.

3. *Hypertrophy of the right ventricle* will arise from impediment and over-filling.

L. Stenosis of the right auriculo-ventricular orifice cannot be diagnosed.

M. Insufficiency of the tricuspid valve offers the following symptoms:

1. *Systolic murmur over the tricuspid valve.* The pulmonary sounds are usually very feeble.

2. *Dilatation of the right auricle* produced by over-filling with blood from the venæ cavæ, and from the ventricle by regurgitation during its systole.

3. Venous pulse and hepatic vein pulsation.

FUNCTIONAL DERANGEMENT OF THE HEART.

The heart, as the central organ of circulation, will become functionally deranged, not only when its own circulation or nutrition is disturbed, but also, and most frequently, from disturbances of circulation and nutrition in any important organ of the body, especially when such disturbance is of any considerable duration.

Symptoms of functional derangement of the heart differ but very little from such as are manifested in actual heart lesions, or tangible anatomical alterations in the tissues of the organ.

The disturbances may be either in the quantity of heart force: systolic or diastolic action, or in the quality: rhythm, sound, or in both at the same time.

As in anatomical lesions only the physical symptoms are of any real clinical value, so in functional derangement of the heart, only the objective symptoms will be useful for diagnostic purposes, although, in either case, the subjective feeling of the patient, such as pain, feeling of uneasiness and nerve phenomena, may often be of sufficient significance to clear up a case of heart disease.

When there is a general disturbance of circulation in the body, for instance, in fevers, or in high-graded local inflammations, provided the nature of the disturbance be of such a kind as to impair the circulation or the nutrition of the heart, there will be symptoms showing a reduction or temporary augmentation of the contraction or dilatation of the ventricles or the auricles. The heart pulse and the apex-beat will be more or less modified in intensity, duration or caliber. Correspondingly the pulse of the large blood vessel will show changes, indicative of the cardiac disturbances. When there is insufficient innervation in the tissues, or when there is too great irritability of the highly-sensitive endocardial structure, whether of inflammatory origin or not, the valves become more or less incompetent to close the arterial or venous orifices, and the sounds will at once show alteration in pitch and quality. The following synopsis of symptomatology will convey, in short, the physical diagnosis of functional disturbances of the heart.

Meyer G. (*Heilbare Formen, etc., Aachen*) describes the lightest forms of functional insufficiency, found in young people, who develop too fast up to puberty, and afterward become paralyzed. This insufficiency ceases after complete maturity and total development of the muscular tissue. *Parenchymatous disturbance* after typhoid fevers, diphtheria, weakened heart and syphilitic diseases.

Ricgel (*deutsch. Archiv. fur clin. Medic. XXVIII*) describes *hemisystole* of the ventricles in a young woman. There was a tripartite apex-beat, a tripartite venous pulse and but a single pulse of the arteries. He ascribes it to atheromatous condition of the arteries of the heart.

Malbranc (*deutsch. Arch. fur clin. Medic.*) considers *hemisystole* as a contraction of only *one* ventricle, due to lack of force of the other. It constitutes a symptom indicating great danger, if there exists at the same time valvular trouble.

Francois Frank (*Gazz. des hop.*) finds frustrated or incomplete systole of one ventricle to cause the following manifestations: when there is mitral regurgitation during a ventricular contraction, which is insufficient to produce a full arterial pulse, or when there is exceeding high pressure in the aorta and the blood regurgitates during ventricular systole into the auricle; in either case there will be intermission in the arterial pulse. The next following ventricular systole will be of an unusually great strength, to compensate the former feeble one, and a new aortic tension will follow, and of course a regurgitation and an intermittent arterial pulse, etc.

Premature ventricular systole, when the ventricle contracts before its cavity is perfectly filled; here an arterial pulse, but feeble one, will ensue.

Abortive or insufficient systole, when the *cardiac* pressure is no greater than the *aortic*. An intermission, or a very feeble, alternating with a full pulse will follow. Hemisystole is then either an incomplete contraction of the left ventricle or a total absence of it, *at times.*

Bernheim and Renou of Lyon consider a long-standing condition of this kind a very dangerous symptom of alteration of the heart tissues, although not anatomically demonstrable.

Garcin (*Archiv. Gen. de Med.*) treats of functional or relative insufficiency of the mitral valve as follows: "There may be two immediate causes for such disturbances: 1. An actual structural change of the papillary muscles. 2. Dilatation of the ventricles, causing widening of the auriculo-ventricular opening to an excessive extent. The distant cause may be over-fatigue of the heart's tissue. Such conditions exist in people who carry heavy burdens up hill or up steps, or make long and forcible marches. If not of too long standing, and not too severe, they are often curable. The symptoms in the early stages are: palpitation, shortness of breath, pain in the chest, vertigo, sometimes hemisystole without tangible anatomical lesion. In the second period of this malady there is permanent ventricular dilatation, low position of the heart in the thorax, displacement of the apex-beat and an extensive area of dull cardiac sound. In the third stage, there is *mitral* insufficiency; very seldom *tricuspid.*

The fourth and last stage is characterized by *cyanois* and *dropsy.*

Perfect insufficiency may be caused: 1. By disturbance of circulation, from aortic derangement, endocarditis, hypertrophy of the left ventricle or atrophy of the kidneys, emphysema, interstitial pneumonia, with dilatation of the right ventricle. 2. By paresis of the muscular tissue in endocarditis or pericarditis, in consequence of nervous derangement of the heart, hysterical trouble in the nervous centers, typhoid, severe exanthematous and puerperal fevers, cachexia, severe malarial troubles, chlorosis, icterus of graver forms; chronic and acute poisoning, alcholism, etc.

The difference between relative and absolute insufficiency is the amount of force in the murmur, which is greater and more constant in the latter than in the former.

In relative insufficiency the pulse is nearly always regular, in absolute almost ever irregular. In relative it is full; in absolute small and feeble. General disturbance of nutrition of the body takes place much sooner in absolute than in relative insufficiency.

Nothnagel (*deutsch. Archiv. fur clin. Med.*) speaks of Arythmia as being a deviation of the regularity of the heart pulse, and continued into the arteries as irregular arterial pulse. There are four different forms of it. 1. Pulsus bigeminus and alternans. 2. Periodical irregularities, that is, the duration of the pulse is variable. 3. Irregularity of frequency. 4. Intermittent, or irregularity in the interval between the individual pulses. The causes of Arythmia he considers to be disturbances of function or lesions of the heart ganglia, of the cardiac filaments of the pneumo-gastric directly, or by reflex action from sensory fibres of the cardiac nerves; also disturbance of the centers of the pneumogastric. It is most pronounced in general atony and anaemia.

Jouane, Leyden and Potain describe duplication or double pulse, which they ascribe to general nerve derangements.

Table II.

Aneurism of the arch of the aorta, forming a number of sacs. One protruding through a perforated portion of the sternum near the right sterno-clavicular articulation.

Case *R.*—A man of sixty-eight years, of a strong constitution, but very dissipated habits.

History.—Two years before his death, he consulted Dr. Dumenil about a large tumor on the right upper portion of the sternum. An aneurism of the arch of the aorta was diagnosed and the patient advised to lead a more regular life. This advice he did not follow. About two weeks prior to his death he was brought to the hospital in the following condition:

Symptoms.—The tumor had not changed in size from two years ago; the man was exceedingly emaciated, and the slightest movement he made was very painful, and produced suffocation. He was occasionally attacked with fits of fainting, and very profuse perspiration. He had a violent cough, and expectorated some mucous. The pulse thin and very slow. Lies on the right side, and looks gloomy. The stethoscope placed over the tumor reveals a double pulse synchronous with the apex-beat. The heart is pushed down below the sternum. On the left side respiration is normal; on the right, bronchial respiration and bronchophony in several places. The next day a fainting fit, but not of such duration as the previous day. During the fit the pulse is fluttering and scarcely perceptible, the action of the heart tremulous and uncertain. Three days later he was taken with a violent rigor, which lasted several hours. The rigors returned for several days at exactly the same time of the day, and were stopped by Sulph. Quinia in sufficient quantities. The pulse after this became more and more frequent, and the heart-action more stormy. Respiration became almost impossible. The color of the face became livid. Died fifteen days after admission to the hospital.

Post-mortem.–Externally the tumor is of ovoid form, and about two inches in diameter and one high. The skin over it is normal. Internally it was lodged under the sternum, and gave origin to the subclavian, vertebral and common carotid arteries on the left, the innominate artery on the right. The cavity of the left subclavian vein, which crossed obliquely the posterior surface of the tumor, was closed up by a brown thrombus. A large vein entered the right subclavian and took the place of the left. Below the origin of the left subclavian, vertebral and carotid arteries, a bifid aneurismal sac was found, which was smaller than the above. The left lung was extensively excavated to lodge the tumor, was inseparably attached to it; its tissues below the sac were obliterated. The trachea, opened from behind, showed that the tumor has driven its anterior wall backward, and very nearly closed up its cavity. The mucous membrane at the bifurcation was corroded. The pneumogastric nerve was compressed and flattened, and in some portions of its course was obliterated. Portions of it were sclerotic.

Fig. 1 shows in natural size the details of the aneurismal sac opened from behind. Above the aortic orifice (*A. O.*) there was an enormous spherical enlargement (*A. D.*). The inner wall of this vast sac was covered with calcareous plates (*A. C.*). An incomplete fibrous ring or seam marked the termination of the sac on each extremity. From this sac originated the large aneurismal tumor (*C. C., T. A.*) and two small ones, one of which protruded into the pericardium, and the other into the trachea, and pushed it backward. All the sacs were filled with clotted blood. The larger tumor, which originated between the right innominate and carotid arteries, was divided into two parts, one substernal and the other peristernal, but both communicated with this bone (Fig. 1 gives a vertical section of it). The sac was full of clots, disposed in concentric layers around a number of central nuclei. The layers were of unequal density. The solidest were near the vascular wall; and all seemed to have had free communication with the circulating blood. There was no trace of any vessels in any portion of the clots and neoformations. The colors of the several layers and parts varied from a perfect white to a deep reddish brown and yellow. The left common carotid originated in the aneurismal pocket, and was occluded by clots and other solid matter, whilst the left subclavian, vertebral and the right innominate arteries were open. The great dilatation (*A. D.*) was covered on its inner surface with calcareous, cartilaginous and osseous plaques of different sizes and dimensions. All three coats of the vessel were involved in the dilatation. The middle and inner coats of the smaller tumors were destroyed, but were replaced by a dense fibrous structure, and perforated in many places. The outer coat still existed, but was reduced to an extremely fine membrane. The portion close to the sternum was broken through, and the blood was allowed to communicate with the peristernal connective tissue. The whole inferior lobe of the right lung was covered with innumerable miliary abscesses. A great quantity of solid tuberculous matter was scattered through the organ.

Figs. 2, 3.–An individual died suddenly of very exhaustive hæmorrhage, and apparently without any disease-premonitions. The post mortem showed the descending portion of the arch of the aorta to be of unequal diameter, wrinkled and covered internally with projecting plaques (*A. P.*, Fig 2); below the alteration a small cavity (*P.*) leading directly into the œsophagus (*O.E.*). When the œsophagus (3) was opened, it was found that a large orifice was formed in its walls by the vessel. A number of similar perforations were formed into the left bronchus (*P. O. E.*) (*B. G. P.*). and (*B. D.*) right bronchus.

ALTERATION IN SOUND PHENOMENA.

Nixon (*Dublin Jour. of Med. Science, Sept. 1881, No. 95,*) describes functional murmurs of the pulmonary artery, which are due (independent of anaemia, or direct pressure upon the trunk or branches of this vessel) to the following derangements:

1. Acute rheumatism.
2. Typhus abdominalis.
3. Continued fevers with profuse perspiration.
4. Bronchitis or œdema of the upper lobes of both lungs.
5. Paraplegia from general myelitis, tubercular meningitis (cerebral), hysteria and hypochondria.
6. Considerable upward pressure of the diaphragm, such as occurs in extensive abdominal serous effusion.
7. Long-standing diarrhœa, dyspepsia.

The sound is of *the friction kind,* and is, as a rule, very transitory.

At one time greater importance was attached to it than it deserved.

Sansom (*Med. Times*) *remarks on the* causes and significance of duplicate heart-sound, that it may only be fictitious, that is, it may be due to too great tension of the flaps of the mitral valve during *auricular* systole, or may be real, that is, be caused by tension of one flap more than the other during *ventricular* systole.

Fraenzel (*Zeitsch. fur clin. Med.*) describes *galloping rhythm* (*gallop rhythmus*) as being a triple tone or sound of the heart's action, two sounds occurring in diastole, one in systole. It is similar to the stroke of a horse's hoof, in a gallop, upon hard ground, heard from a distance. It is audible over the whole cardiac region, and occurs in ileothyphus, exanthematic typhoid, grave forms of pneumonia and many other diseases, excessive weakness of the heart; and although only functional, it forebodes great danger to the patient.

Lepine (*Lyon medic.*) found a double apex-beat in gallop rhythm. The tones were presystolic, and indicated excessive ventricular dilatation.

Lepine (*Gazz. des hopit.*) *states that the bruit de galop* consists of *two short sounds* followed by a long one ∪∪ —. In time they correspond to the auricular systole and ventricular presystole. It is rather muffled, and may be considered rather a stroke than a tone.

The diseases in which he found it present were: 1. Simple cardiac hypertrophy, consequent upon nephritis.

2. Dilatation of the right ventricle, consequent upon gastric dilatation with diseases of the liver. In both conditions there is great arterial tension, in the first class, in the *aortic* system, in the second class, in the *pulmonary* artery.

This excessive arterial tension causes lessening of the venous tension and increased activity of the auricle, which becomes hypertrophied. A sudden strong contraction of such an auricle produces the *Bruit de galop.*

In no other diseases are these conditions existing, except those described, and in stenosis of the left auriculo-ventricular orifice, when the auricle meets with a similar resistance; and then also the *Bruit de galop* is produced, and added to it is the blowing murmur of a narrow orifice.

Morel, V. (*in Recherches experim.*) found that diseases of the abdominal organs will produce, sometimes, permanent functional disturbances of the heart with the following symptoms.

1. An increased strength of the second heart sound in the pulmonary artery.
2. A duplication of the second sound.
3. Murmur in the tricuspid and bicuspid valves.
4. Murmur in the right heart, with a true venous pulse in the neck and liver. This abnormity is due to resistance in the pulmonic circulation, brought about by reflection from the abdominal sympathetic ganglia upon the cervical medulla, and from here upon the nerves derived from the cardiac plexus, and connected with the cardiac ganglia.

Widmann (*Allg. Wiener med. Zeit. 41*) in speaking of *bruit* without any existence of valvular lesion, and on the other hand of valvular lesions not manifested by bruits or murmurs, states, that bruits exist in stenosis of cardiac orifices when the blood current has a certain amount of force and velocity; also that the orifice through which it is to pass must be altered to a certain extent. Accordingly, there will oftener be absence of murmur in stenosis of the auriculo-ventricular openings than in valvular incompetency; and the greater the constriction of an orifice, the more distinct will be the murmur; in insufficiency, on the other hand, the more considerable, the weaker the bruit, for here the resistance to the blood will be least. In chlorosis and leukæmia the causes of the murmur are insufficiency of the valves from weakness or loss of

ANEURISM OF THE ARCH OF THE AORTA. **Sec. IV.** *Tab. II.*

Fig. 1.te

Fig 2

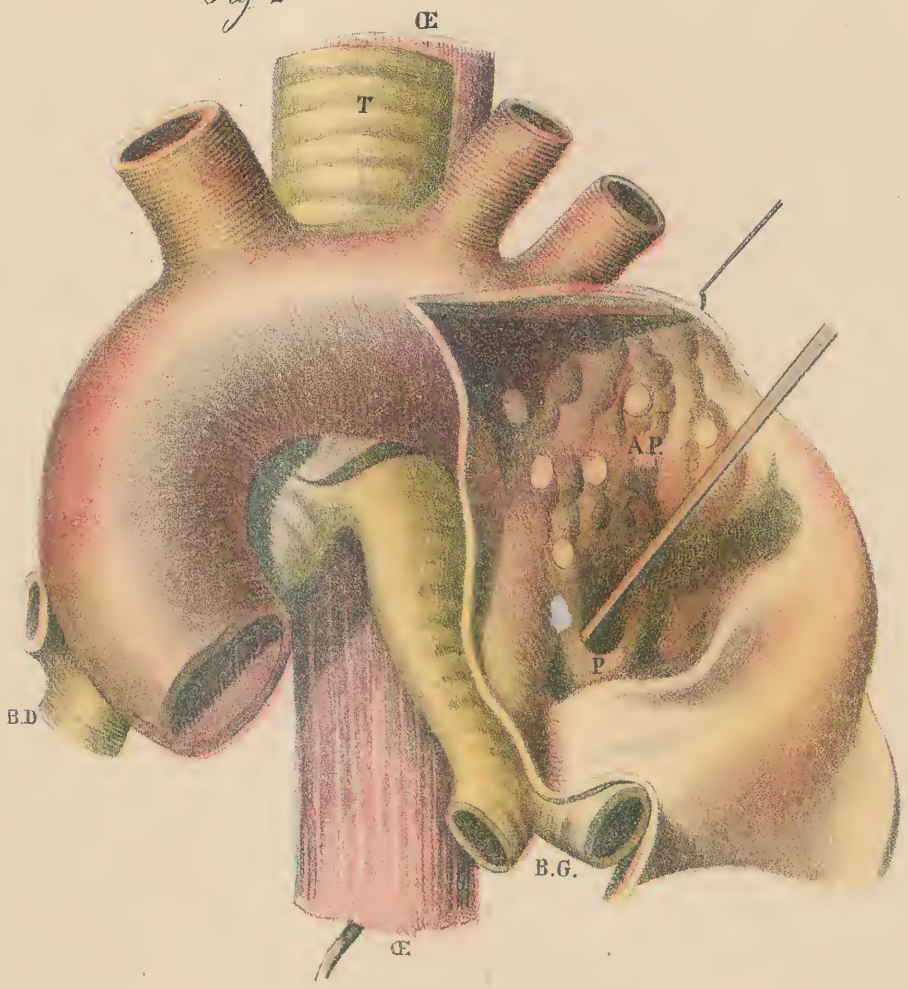
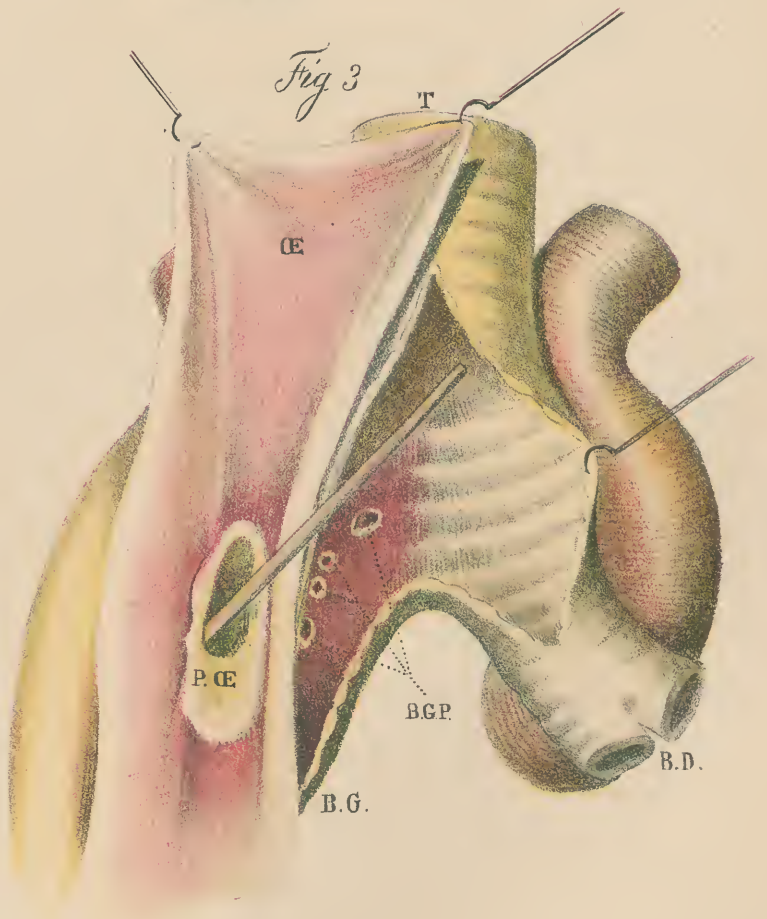


Fig 3



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Sec. IV. *Tab. III.*

ANEURISM OF THE THORACIC AORTA.

Fig. 1.

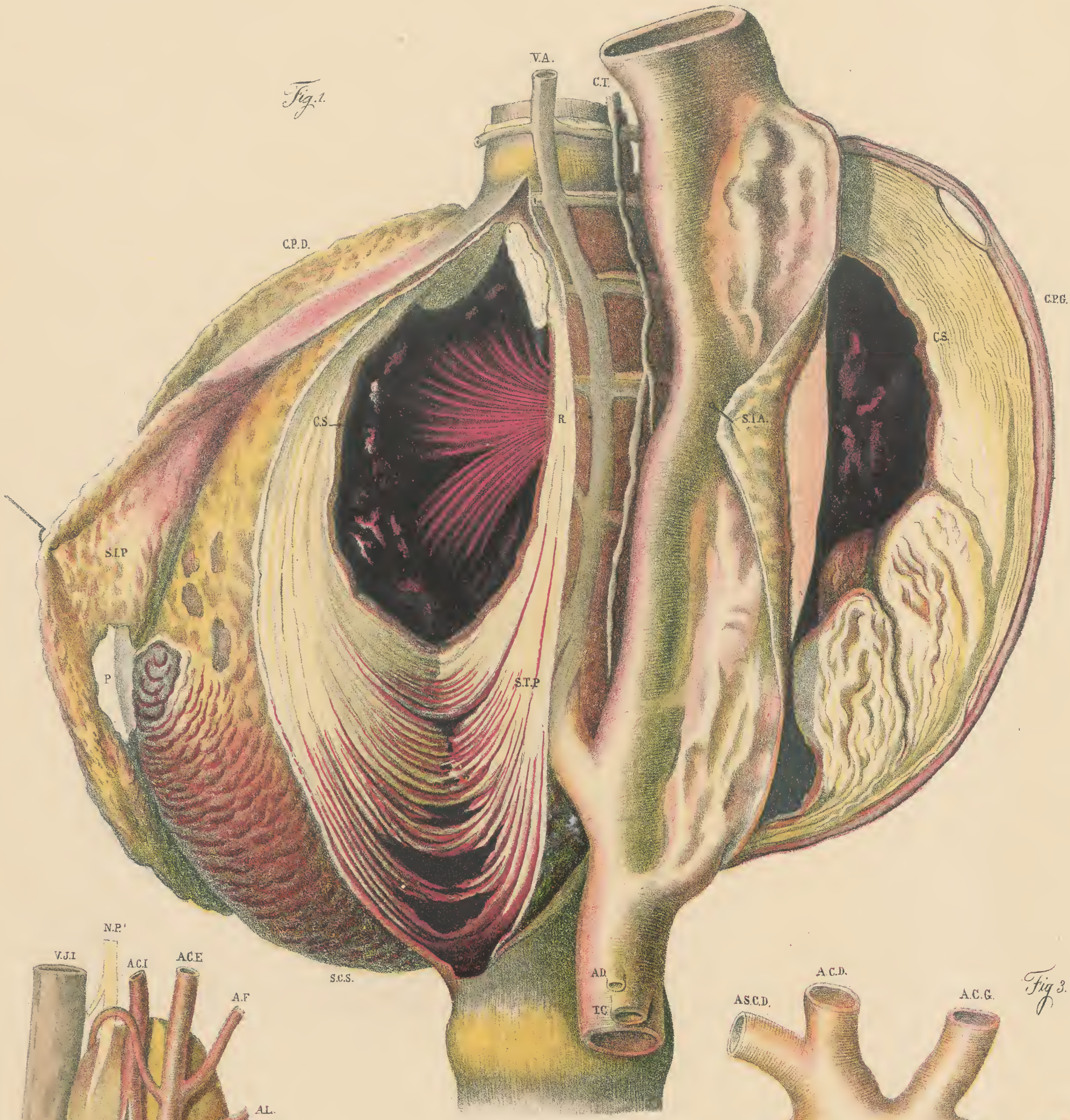

Fig. 2.

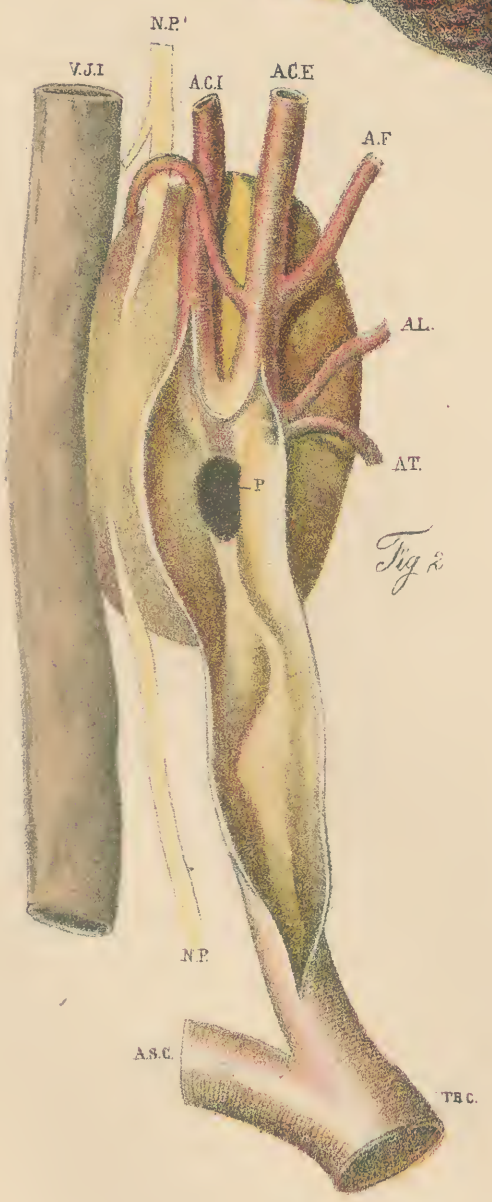

Fig. 2.

Fig. 3.

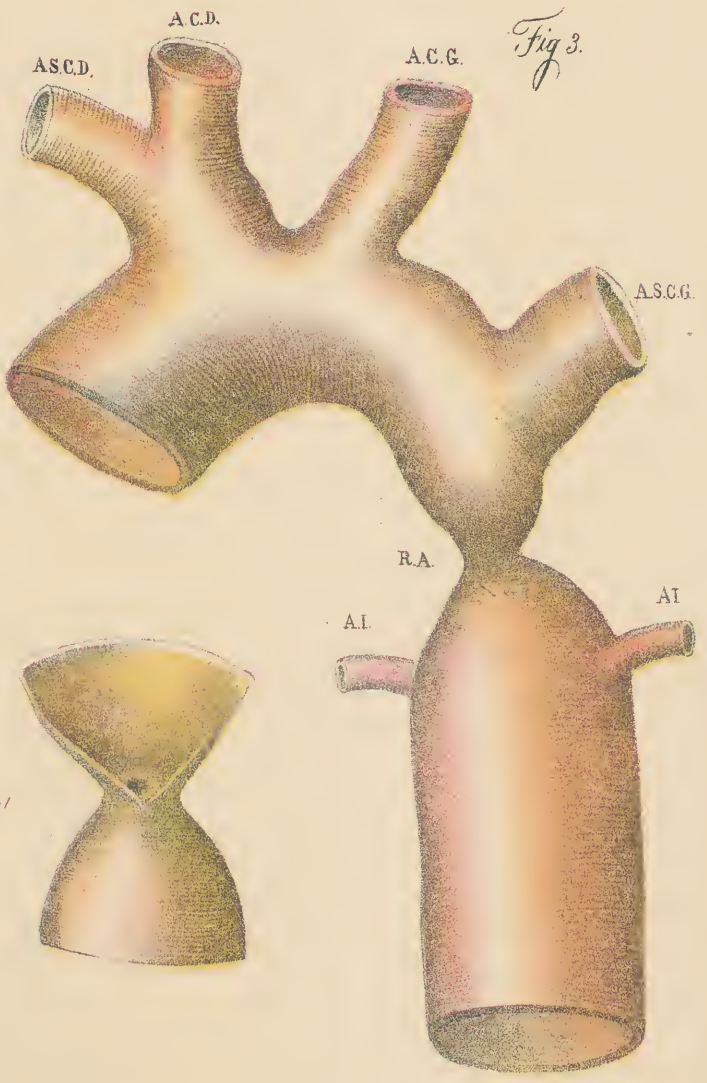

Fig. 3'

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TABLE III.

ANEURISM OF THE THORACIC AORTA.

CASE.—A man of forty-one years, at the charity hospital.

Subjective symptoms as described by the patient himself: "a sense of very painful constriction and strangulation, without any intermission, existing in the region of the last ribs".

Objective symptoms: Face yellow and waxy appearance; respiration hurried; suffocation on the slightest bodily movement, also enormously profuse perspiration; pulse barely perceptible; cold extremities.

Percussion of chest: In front, perfectly clear sound on *the left upper two-thirds*; on the right upper third of the thorax, the same; on lower third absolute dullness right and left; laterally and posteriorly, total dullness all over.

Palpation: Very distinctly palpable beating in the whole anterior portion of the chest; is also distinctly visible; the beating constitutes a sudden and strong jerk, which lifts up the whole side of the chest, and is connected with a peculiar push from left to right. It resembles a strong apex-beat of a highly hypertrophied heart, and is situated in the right side of the chest.

Auscultation: No murmur; upper part of chest a clattering bruit; a double beating pulse, increasing in intensity downward toward the region of the liver, where the jerking beat is strongest.

He lived in this condition twenty-five days. Died suddenly on the twenty-sixth day after admission.

Post mortem: An enormous tumor filling up two-thirds of the chest cavity; recent pericarditis; very dilated heart, flattened in a postero-anterior direction; both lungs compressed, and reduced to one-third of their former size; pulmonic tissue soft and infiltrated; and the bronchial mucous membrane covered with tubercles.

The aneurismal tumor was single, but consisted of two distinct lobes, both connected with the aorta in front and the vertebral column behind; the base of the right lung solidly adherent to the tumor; the larger azygos vein (*G. V. A.*) and the right and left aortic intercostal arteries (*V. T. A. L.*) were obliterated by pressure from the tumor; the greater and lesser splanchnic nerves (*G. N. S. P.*), (*P. N. S. P.*),on the right side, compressed and reduced to thin ribbons, and totally atrophied; the thoracic duct displaced;

Figs. 1, 1, show the special connection of the tumor with the aorta and the vertebral column; the sac was formed at the expense of the posterior segment of the aorta, to the left; the right and anterior segment was, with the exception of a considerable dilatation, normal and perfectly permeable to the circulation; all three vascular coats were uninjured in the dilated portion, but in the injured portion of the tumor they were totally obliterated and replaced by fibrous structure. This figure shows the sac opened, on each side of the vertebral column by a deep, vertical incision; the stratified lamellæ are exposed to view, and the large orifice by which both pockets communicated. The cavity has all the appearance of furnishing a free communication to the circulating blood with both halves of the tumor, and that the heart, at each ventricular systole, impelled the blood in the sac in a lateral direction. From all appearance the walls of the sac were fast giving way, and a rupture would have taken place, had the patient lived but a short while longer; a perforation was already formed at (*P.*) but effusion prevented by the wall of the base of the lung. This organ showed incipient ulceration and destruction of its tissue, in that locality. The interior of the vast sac had all characteristic appearances usually found in well developed aneurismal sacs.

Figs. 2, 2, represent the innominate (*T. B. C.*), and the right common carotid arteries (opened in front) perforated at (*P.*) and communicating by a large opening with the aneurismal sac, connected with the posterior portion of these vessels, the internal and external carotids (*A. C. I.*, *A. C. E.*), the thyroid (*A. T.*), lingual (*A. L.*), and facial arteries (*F. A.*). The elliptic opening is situated a few lines below the point of division of the common carotid, the sac firmly adherent to this vessel and the internal jugular vein (*V. I. I.*); the pneumogastric was ingrown with the sac and atrophied; the circulation in the artery was uninterrupted. Fig. 2 shows the opened sac exposing the clotted contents; the internal coat of the artery was completely destroyed, but the middle and external were intact.

Figs. 3, 3, present a case of excessive constriction of the aorta almost immediately below the origin of the subclavian (*R. A.*). Fig. 3. shows the exact caliber of the vessel; above and below the stricture there was no change whatever in the vessel; the left innominate, subclavian and carotid arteries were wider than usual, the same was the case with the first aortic intercostal arteries (*A. I. A.*)

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tension of the papillary muscles and the chordæ tendineæ. In consumptive fevers murmurs are due to superinduced anæmia of all the muscles of the body, and of the heart especially.

Angina pectoris is a symptom of functional disturbance of the heart.

V. D. Harris (*Remarks on angina pectoris, St. Bartholomew hosp. Rept.*) gives a synopsis of angina pectoris: "There are two varieties of this malady; they are connected with gout and rheumatism. Their pathological anatomy is not yet quite known. One is always associated with valvular diseases, and especially aortic insufficiency; there is much pain and dyspnœa, a sense of suffocation, but generally without any change in the pulse; and it is liable to produce sudden death. The other is seldom connected with valvular trouble, but mostly with dyspepsia. The pain is not very great, and often intermits. Danger to life there is but little; but this disease is of more frequent occurrence than the first."

Huchard (*Union medicale*) speaks of neurotic angina, which is due to severe irritation or injury of the filaments of the pneumogastric. It is mostly attended with great pain, and often produces dyspnœa. Derangements of liver and abdominal disorders seem to produce this form of angina by reflex irritation.

DISEASES OF THE BLOOD VESSELS.

PATHOLOGICAL ANATOMY.

All blood vessels are essentially larger or smaller endothelial tubes strengthened on the outside by a greater or lesser quantity of muscular and elastic tissue, constituting one or two distinct coverings. The vessels may be affected in *either* of their *walls alone*, in two or three coats; or conjointly with the surrounding tissue. The larger vessels are generally diseased, independent of the parenchyma in which they run; the smaller, and chiefly the smallest, always partake of parenchymatous affections. Besides congenital vascular anomalies of position, origin, size, etc., which may cause disorders in the body to a greater or lesser degree, and may become of considerable clinical importance, *angiopathology* considers mainly acquired disorders or lesions of the blood vessels.

Simple atrophy—Degenerative atrophy—Infiltrated state of the vessels.

General marasmus of the body is conditioned by a simple atrophic state of most of the smaller blood vessels, the same as in simple atrophy of an organ. When a limb is amputated, the vessels of the stump shrivel up and atrophy from disuse.

Atrophic portions of a vessel, or only a part of one of its coats (the muscular most frequently), are very commonly found in inflamed or otherwise degenerated states of vascular tubes. Fatty alterations are the most frequent in the inner and middle coats, very seldom in the external. The *intima* of the aorta and of the pulmonary artery is most commonly the seat of fatty change; the same is the case with the inner coats of larger arterial trunks. Small fatty spots are not perceptible to the naked eye; larger ones have an opaque white or yellowish appearance. At first the surface is smooth, gradually it becomes rough and velvety.

The beginning of the process consists of the filling up of the cells with fat globules. In the further progress of the disease the fat molecules not only occupy the place of the cells themselves, but also infiltrate into the intercellular spaces. Soon emigration of round cells from the vessels takes place, they absorb the debris left of the destroyed cells, and fatty granulated cells are formed from them.

In the aorta the elective places of fatty alterations are close to the semilunar valves, and near the origin of the arterial trunks; it chiefly takes place in old age and in anaemic persons. When the blood becomes stagnant in the cardio-pulmonic circuit, and the carbonic acid is not sufficiently eliminated from it, then fatty change takes place in the pulmonary artery. The same is the case in the veins, by haematic stasis. In the capillaries fatty degeneration is brought about by disturbance of circulation, usually existing in septic and infectious diseases.

Fatty change of the muscular coat of the vessels is more dangerous than that of the endothelium, for extensive and fatal haemorrhages take place when any considerable quantity of that tissue is altered. Besides, infiltration with calcareous matter and its substitution for the muscular tissue may take place to such an extent as to cause rigidity of the vessel, and the circulation therein will become greatly impaired. Fatty degeneration of the external coat, whether in the vessel alone or associated with the surrounding tissue, if not very extensive, is seldom dangerous to the general health. The afferent lymphatics often deposit quite a quantity of fat upon the external surface of vessels without producing any particular injury. (*Virchow, Langhans.*)

Amyloid degeneration is nearly as frequent a vascular lesion as the fatty change. In the larger vessels the inner coat, of the smaller and smallest the inner and middle coats are usually altered.

There are two forms of hyaline degeneration, one taking place in the inner coat of the larger vessels, and consisting of a change into a homogeneous connective tissue, containing but few cells. The early stages of atheroma of the blood vessels are of this nature. The other form is found in small vessels, forming very close and copious networks, such as the glomeruli in the kidneys and the choroid membrane in the brain. A nuclear proliferation always precedes these derangements. They often form small nodules on the outer coat of the capillaries, and frequently produce thrombi within.

Calcification of the vascular walls takes place when the nutrition of the vessel is impaired, and is generally associated with fatty

TABLE IV.

ATHEROMA OF PORTIONS OF THE AORTA AND OTHER ARTERIAL TRUNKS.

Fig. 1 represents the interior surface of the aorta, opened posteriorly. The whole length of the vessel is covered by a vast number of cretaceous yellowish and whitish plaques, some having a free surface, some covered with a very thin membrane; in the cavity of the aortic sinus, where the coronary arteries originate, on the semi-lunar valves (*V. S., V. S.*), there are great quantities of incrustations of phosphate of lime; the valves are slightly verted; the internal tissue is normal, and is continuous with that of the mitral valves (*V. M., V. M.*), the orifices of the left innominate, common carotid and the subclavian arteries (*O. I., B. C., O. A. C., O. A., S. C.*). The section of the artery was made in such a way as to show, in its whole extent, all the aneurismal tumors or dilatations marked 1, 1. The internal coat of the vessel remained intact, whilst the middle and external coats were perforated by the tumors, which protruded upon the outer surface of the vessel; the internal coat had undergone repair in those places where fissures originally existed, and through which the blood passed into, between and outside of the vascular tunics.

Nos. 2, 3 correspond to the projections formed upon the external surface of the vessel. No. 5 designates a clot situated at the orifice of the inferior mesenteric artery. An incision into the superior mesenteric artery (*A. M. S.*) shows the freedom of the cavity from clots, etc., down to the aneurismal enlargement. Most of the branches of this vessel were occluded by clots at their origins, and collateral circulation established.

Fig 2 indicates a spheroidal aneurism of the right vertebral artery (*A. A. V.*) at the anterior pyramids.

Fig. 3 shows the stratified contents of the opened sac.

Fig. 4 represents the anterior surface of the medulla oblongata considerably depressed by the tumor; the color of the pyramid is changed into a yellowish brown, and partly bright yellow, showing the existence of former hæmorrhages into the brain.

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change, or with sclerosis and atheroma. The lime deposit takes place in the atheromatous or sclerotic parts of the tissue of the inner and middle coats, and often plates and cylinders of lime are thus formed.

Portions of large or middle-sized arteries are converted into solid tubes by this process. As a rule, the muscular coat is thus altered. In heteroplastic formation of the central nervous system the capillaries are found to have undergone calcareous change. The veins are more rarely affected than the arteries, but in the varices they often form the inner portion of the destructive process of the thrombi.

Inflammation of the blood vessels, Arteriitis and Phlebitis.

There are two principal forms of morbid alterations, produced by inflammatory processes in the vascular walls. One is the kind taking place in every inflammation of the bodily tissues, when the vascular walls become more permeable and the mutual exchange of the material between the blood and the tissues is essentially altered.

The other is the strictly angiopathic change of the wall alone, in consequence of the presence of foreign bodies, (the thrombus) in the vessels, which leads to formation of granular and cicatricial tissue on the inner surface of the artery or vein. This latter form may be designated as *plastic* and *obliterating endoarteriitis* or *phlebitis*.

There are, besides, a number of forms of inflammation going on in the vascular walls, which leave behind greater or lesser alterations of a temporary or permanent character, in the whole wall, or in parts of it.

Purulent arteriitis takes place most frequently in suppurating wounds or ulcers. The arteries undergo a suppurative process and decay. The start of the morbid condition comes from suppurating tissue, and begins in the outer coat and gradually passes through the other coats. The vascular wall in purulent arteriitis has a yellowish or grayish white color, and is thickened. Very often the coats break through, and hæmorrhage takes place to a greater or less extent, for instance, in secondary hæmorrhages of wounds, in ulcerated lungs in phthisis, ulcerative carcinoma, etc.

Purulent inflammation of the umbilical cord in new-born babes often produces fatal hæmorrhages. The invasion of microorganisms into a wound seems to be the most frequent cause of such arteriitis. Now and then it may be produced by phlogogenic substances circulating in the vessels, when the purulent thrombi are developed in ulcerated surfaces of the intestinal tract, and from there carried into the venæ portæ by the capillaries. (Dysentery). Occasionally they originate in suppurating wounds. By the passage of the thrombotic masses into the blood-current, pyæmia and formation of metastatic abscesses are brought about.

Hyperplastic arteriitis.

Acute aortitis is an inflammatory process, in form and consequences similar to *endocarditis* and *myocarditis*. In the inflamed portions the surface looks turbid and softened, or there are in it grayish translucent patches, or other excrescences, or fibrinous floculi, very much like those seen in endocarditis. In the external coat there are grayish red foci, generally but little perceptible by the naked eye.

Under the microscope, in the affected portions, all the coats of the vessel are found to be involved. The vasa vasorum are turgid, with round cells and granular or fibrinous masses.

Necrobiotic arteriitis may be caused by infectious *emboli*. When the inflammation and the gangrene pass into the adjacent tissues, abscesses are formed there. The pus corpuscles collected in the vascular wall originate in the vasa vasorum, or in the adjoining blood vessels.

Purulent phlebitis is found under similar circumstances, and the morbid appearance of these blood vessels is nearly like that of the arteries. Very frequently their cavities contain fresh or puriform thrombi. Coagulation of the blood in the veins either precedes or follows inflammation of the wall. Occasionally the phlebitis produces a thrombus, which, under the influence of infectious substances or micrococci, undergoes purulent necrobiosis, and the gangrenous substances intensify the inflammatory process still more. Such thrombotic inflammations of the veins are often met in the portal circulation. The external coat shows infiltration and the surrounding walls a great many nodules. In the middle coat the vasa vasorum are also infiltrated and rather smaller in size. The same is the case with the intima. Grave diffuse aortitis is, as a rule, fatal. In lighter forms, the inflammatory stage passes into the hyperplastic. There is a new formation of connective tissue and capillaries in the partly destroyed tissue. In very light inflamation, absorption of the detritus of the altered tissue, and reformation of connective tissue from fibroblasts takes place. (*Koster Pathogenese des endoarteriitis, 1871. Strogonow, Recherche, etc. Archive de physiol. norm. &, pathol. 1876. Zahn, Virchow Arch. 72, vol., etc.*

Like the inflammatory processes going on in the heart and its membranes, there may be periarteriitis, mesoarteriitis and endoarteriitis. All three may take place at the same time in the vessel. When the endoarterial inflammation becomes chronic, there is formed in the internal membrane a quantity of connective tissue, which presents a number of more or less regularly arranged prominences upon its internal surface, which often occupy a great part of the interior of the vessel. The newly formed tissue is sometimes very solid, and then contains blood vessels. Sometimes it is soft and flabby. In either case the normal tissue of the intima is destroyed. This condition of hyperplasia is called *Arterio-sclerosis*. It may exist in smaller as well as in larger vessels, and generally leads to occlusion of the cavity. It has therefore the name of *arteriitis obliterans* added to the above. Generally, when chronic arteriitis has once set in, it will return at different intervals, and then there will be formed new deposits, and the old will increase in volume. Mesoarteriitis is generally associated with endoarteriitis. The muscular tissue becomes obliterated and is replaced by fibrous structure. The alterations in this lesion are never so extensive as in the former. Periarteriitis leads to condensation and thickening of the external coat. Upon arteries but loosely imbedded in tissue, there are formed large knots or fibrous strings, (Periarteriitis nodosa.)

The veins undergo inflammatory reactions in their several tunics similar to the arteries; only that the inflammations are seldom confined to the vessels alone, and the inner coat seldom becomes thickened to such an extent as the arteries.

Generally the hyperplasia is produced by thrombi in the vessels and the inflammation in the adjoining tissues. (*Kussmaul und Meyer, deutsches Arch. fur klin. Medez.*)

Ætiology of the hyperplastic endoarteriitis, like that of endocarditis, is manifold. Whatever is likely to produce a lesion in any serous, fibrous, or muscular tissue in any portion of the body, is capable of giving rise to inflammation in the vascular walls. In fact, the vessels, as they constantly contain blood, which carries all kinds of impurities and offal of the body, and are besides exposed, to a greater or lesser degree, to mechanical injuries, they are liable to inflame oftener than the heart and its membranes. There are two chief causes which, above any other, give rise to chronic inflammation of the vessels: syphilis and tuberculosis. Heubner was the first to thoroughly study syphilitic arteriitis, which he considers a very frequent occurrence. The vessels are affected, either alone or in conjunction with syphilitic parenchymatous diseases. Vascular affections alone show grayish or whitish thickened spots of different sizes in the inner coat, or thick, tuberculous masses or strings on the outer coat. Histologically they do not differ from ordinary hyperplastic connective tissue. The form of luetic arteriitis occurring in luetic inflammatory centra, shows the vessels surrounded by very copious cellular infiltrations, gummatous granulations or cicatricial fibrous structure; here all coats are involved. The peculiar character of syphilitic arteriitis is the manifold histological changes going on in the vessel, far beyond any found in ordinary arteriitis, and which very frequently lead to occlusion of the vascular cavity, or obliteration of its walls. The veins are as liable to become the seat of gummatous syphilitic inflamation as the arteries. Differential diagnosis of syphilitic and non-syphilitic arteriitis can only be made by thorough examination of all parts of the body and ascertaining the actual presence of syphilitic *gummata* elsewhere besides the place where arteriitis exists. (*Baumgartner, Virchow Archiv. Langenbeck, Archiv fur clin, Med.*)

Inflammation of the vascular walls is a very common occurrence in tuberculous tissue. There may be formed in the wall of a vessel tubercles or more diffuse inflammatory processes. When the formations become caseous, the vessels will also become involved. The affected arteries may then break and more or less copious hæmorrhage will follow. Affected veins may carry the products of their diseased walls into the general circulation and metastatic abcess or *caseous foci* will be formed where such detritus is deposited and remains. The tuberculous process may give rise to fibrinous hyperplasia of the inner or outer walls of vessels. Very extensive hyperplasia may produce obstructions in the vascular cavities, especially when the walls contain tubercular granulation. The vessels may be closed up altogether by thrombi.

Fig. 1.

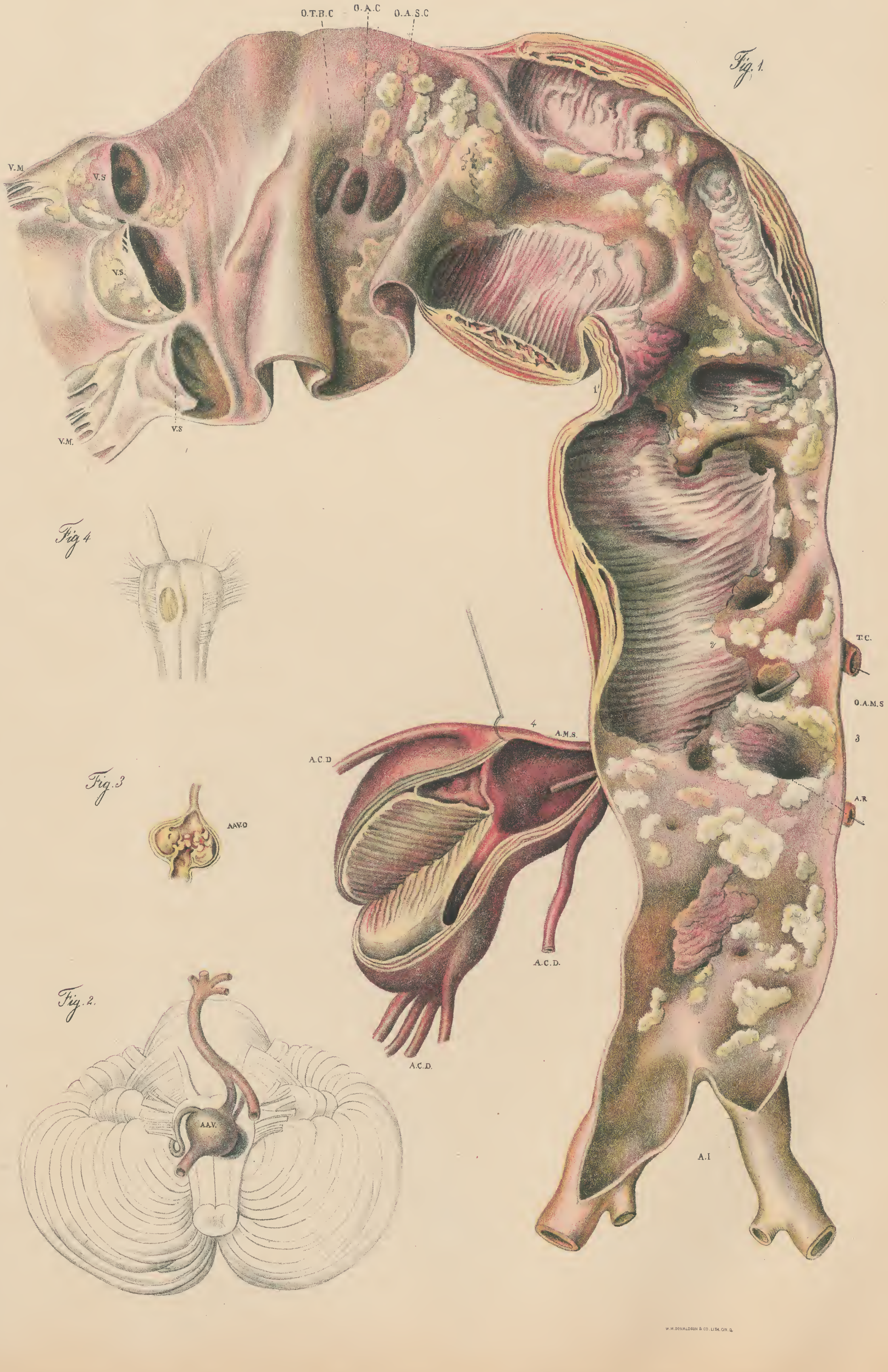

Fig 4

Fig. 3

Fig. 2.

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Sec. IV. *Tab. V.*

PURULENT ENDO-ARTERIITIS.

GANGRENE OF RIGHT FOOT AND LEG.

SCLEROSIS OF THE INNER ARTERIAL COAT.

Fig 4.

Fig 7.

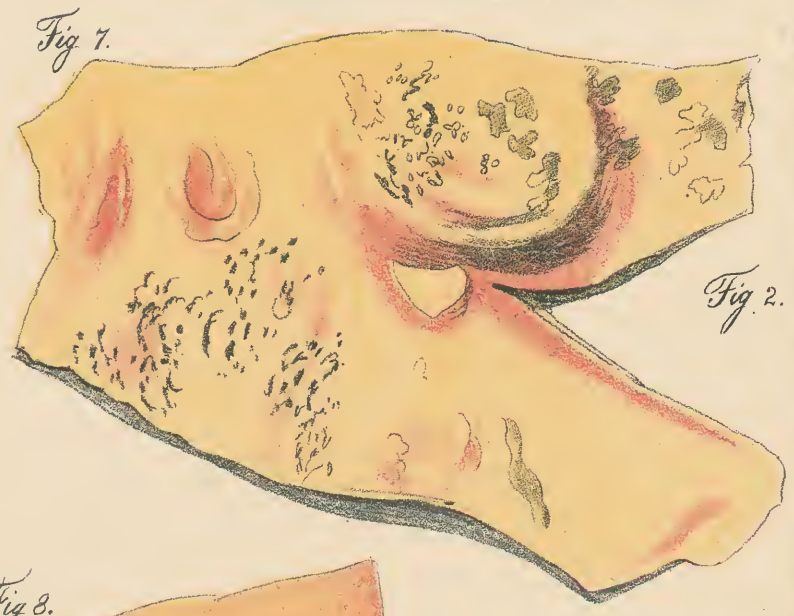

Fig 2.

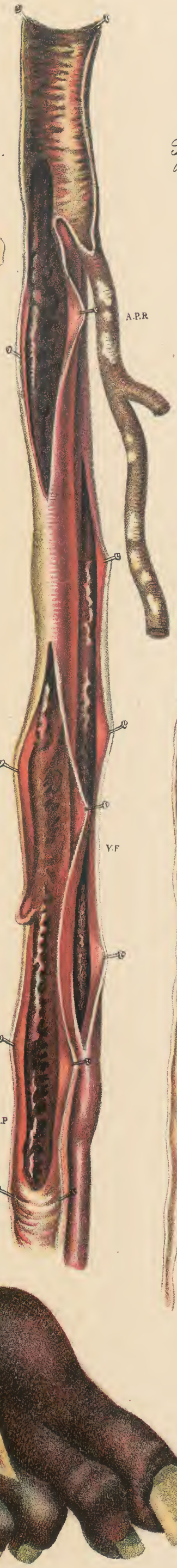

Fig 3

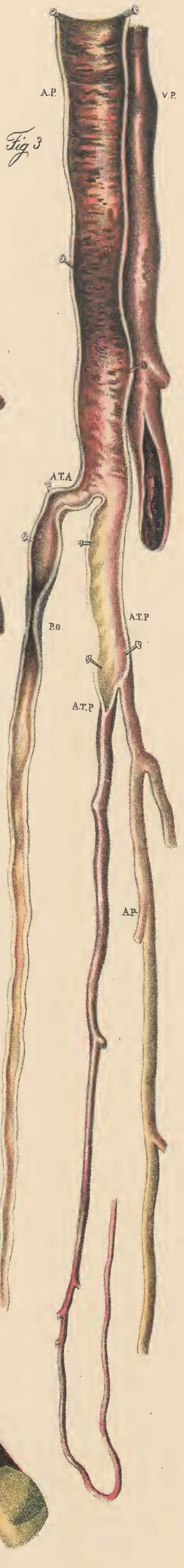

Fig 5

Fig 8.

Fig 6.

Fig 9.

Fig 1.

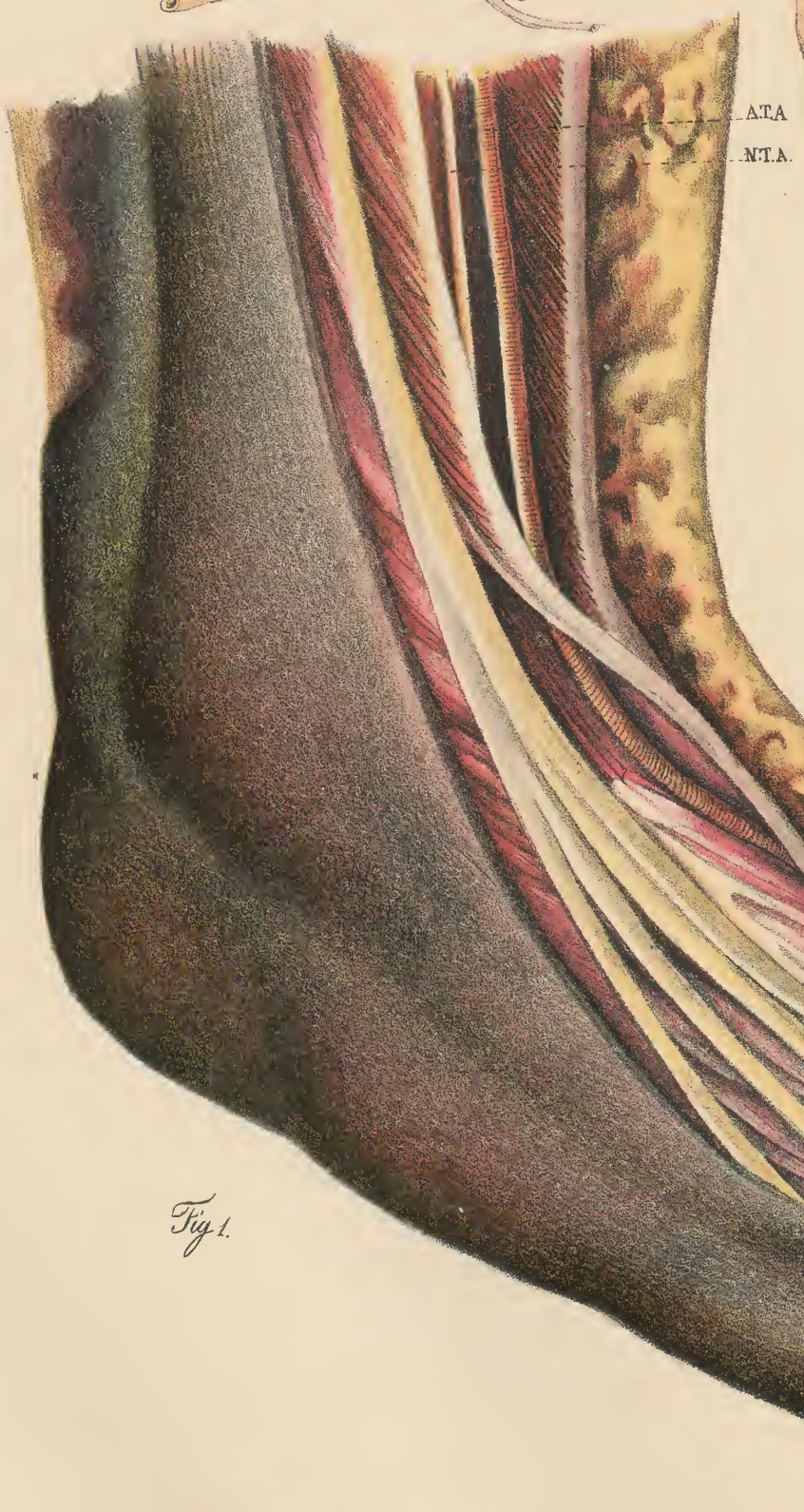

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TABLE V.

Purulent endoarteriitis of the right femoral artery, and its divisions below the profunda, terminating in gangrene of the whole limb.

CASE.—A woman of fifty-eight years.

History.—Was affected for many years with *carcinoma recti*, producing extensive suppurating sinuses into the vaginal cavity, and extending to the cervix uteri.

Symptoms.—She was exceedingly anæmic and very much emaciated. Three days before death she was taken with excruciating pain in the toes of the right foot; it was at first thought that the pain was of neuralgic character, and treated accordingly, but without affording any relief. The next day the pain extended along the course of the arteries of the foot and leg, and gangrene at the toes appeared, which spread with great rapidity upward. Amputation in her condition was out of the question. All means to prevent the spread of the gangrene and afford relief, were used, but without avail. She died the third night after the beginning of appearance of gangrene.

Post mortem.—It was found that the recto-vaginal septum was completely gangrenous. The vessels in that region were obstructed. The uterus was but slightly affected.

The gangrenous foot and leg were black, and the mortification had chiefly affected the anterior and external surfaces, showing posteriorly distinct lines of demarcation. The skin was dry and shriveled, and the tissues, although dead, were not putrid. They emitted an extremely nauseous odor. The muscular structures were still coherent and perfectly distinct. The anterior tibial artery and the anterior tibial nerve were not at all altered exteriorly.

Fig. 1 represents the gangrenous foot and lower portion of the leg. The skin has been dissected off, to show the subcutaneous structures.

Figs. 2, 3. show the femoral artery from a little below the groin, downward. The femoral and popliteal arteries and their branches are filled with calcareous masses, and remain perfectly cylindrical after being cut. The solid masses formed rings within the cavity, and when passed between the fingers, feel like larger branches of bronchi.

The femoral artery (*A. F.*), Fig. 2, from below, the profunda femoris (*A.P.R.*) is filled with many colored clots. The same is the case with the popliteal (*A.P.*), Fig. 2, anterior tibeal (*A.T.A.*), Fig 3, and the trunk of the tibio peroneal artery (*A. T. P.*), Fig. 3. The posterior tibial artery (*A.T.P.*) is exceedingly small. The clots extended to all branches of those vessels. The venæ comites were likewise filled with clots and calcareous masses.

Figs. 4, 5, 7, 8 show the different stages of atheromatous development upon the inner and middle coats of the largest arteries.

Fig 6 shows an obturated and altered artery. *a. b.* hypertrophy of the external coat.

Sclerosis and Atheroma of vessels. Sclerosis of arteries is called that state in which there are local centers of thickening or condensation of the inner coat. There may be formed larger or smaller projections upon its surface, of different forms; they may be transparent, gelatinous, very dense like cartilage, or fibrinous. Such condensations may take place from the origin of the aorta, and its valves at the heart, down to the farthest arteriole. The number of such condensed spots may be very small, or exceedingly great. The intima of the aorta is, occasionally, thoroughly sclerotic, and without a single spot of normal tissue. In such a widespread change there are found gray transparent plates by the side of cartilaginous knots, and yellowish white or clear white plaques. They may have rough or smooth surfaces. Often the mortified portion of the tissues forms ulcers, and contains white detritus of the former cellular structure. Sometimes the rough surfaces are covered by abcesses and decaying thrombi. The yellowish white spots are designated as *atheromata, the defects* as *atheromatous ulcers.* The whole process is the result of the sclerotic alteration. Very frequently lime is deposited upon the altered and mortified spots, which become hard and white, and have the appearance of bone. Formerly such a condition was described as arterial ossification, from which it differs greatly. The veins are not nearly as often affected by sclerosis and atheroma as the arteries.

Condensation and thickening of the inner vascular membrane not only interfere with its physiological function of keeping the blood in a liquid state, but also produce anæmia of its own tissue and that of the internal layers of the middle coat, for both receive their nutrition from the blood in the vessel directly. The anæmia causes fatty degeneration and decay of both. Although a constant reparative process is going on, side by side with the decay, by means of cell-proliferation in the vascular wall, the destruction is going on so much quicker than the repair, that the muscular tissue of the middle coat swiftly disappears, and its place is filled by the atheromatous detritus, which consists of fatty molecules of various sizes, calcareous granules and cholesterin crystals. The inner coat, although primarily affected, resists the destruction much longer, and remains intact, until the pressure of the middle coat, growing stronger, finally breaks it through, and the atheromatous detritus is emptied into the cavity of the vessel. A portion of the debris is swept into the circulation, mingling with the blood and producing thrombi in smaller vessels, which eventually are closed up by emboli; a portion adheres to the edges of the fissure, and causes a roughness of the inner vascular surface upon which fibrin is precipitated by the obstacles created by the calcareous and cholesterin masses, to the free movement of the blood. Finally, the fibrin and other atheromatous material fill up the vascular cavity more and more, and call forth, by stagnation of the blood they produce, new morbid processes, such as endoarteriitis, fatty and granular degeneration, etc. The cavity left by the discharge of the atheromatous substances, in the vascular wall, is filled with pus, and its walls undergo ulceration (atheromatous ulcers). Gangrene and other necrotic destructions soon take place in the ulcer, especially when microorganisms are brought into it from the circulation (which happens very often), and new dangers to the vascular tissue are created. Very often infectious thrombi are then produced, which, when carried into the circulation, become a source of multiple metastatic abscesses in the body. In small vessels atheromatous processes, thrombi, and fibrinous deposits cause a complete closing of the cavity, and they are converted into solid cylinders. In larger vessels, the cavities are not perfectly obliterated, some circulation may still be carried on, but finally the blood pressure upon the partly diseased walls causes them to give way in places; pockets and enlargements or dilatations of one or all of its tunics will ensue. As a rule, all the coats blend by adhesive inflammation into one histologically modified membrane. The fate of tissues in which such a process is going on is variable. When collateral circulation is established between the still sound part of the vessel and any anastomotic branch of a near vessel, repair and partial recovery take place. Generally the newly-formed structure is weak, and has the character of cicatricial connective tissue of a low grade of vitality. But when such an atheromatous change befalls a terminal branch of an artery, as in the brain, the liver, etc., then no *restitutio ad integrum* ever takes place. The tissues supplied by such a vessel are hopelessly lost, for very soon anæmia and gangrene will overtake them.

A slower but no less dangerous form of vascular lesion, caused by atheroma and sclerosis, is the formation of thrombi in the cavity of vessels; for, when a portion of the thrombus is swept from it into the general circulation emboli will fill up the vessels which they enter, especially if the vessels be small. Larger vessels may be closed up by enormous hypertrophy of the inner wall, caused by plastic endoarteriitis which the thrombi call forth. (*Voisin, Etudes Clin. Union Medic, No. 86, 101, 131, 151, 1869.*) (*Johnston, Dublin Jour., XLIV, 1867.*) (*Heubner, Arch. f. Heilk, 3, H. 1870.*) (*Langhans, Virch. Archiv. 36.*) (*Koster, Virch. Jahresbericht, 1875.*) (*Thierfelder Atlas der Pathol. Histol., 6, 1881*).

According to all observations and clinical experience, rheumatism, gout, chronic alcoholism, syphilis and tuberculosis are the chief causes of the sclerotic process of the intima. (*Giovanni, Arch. Ital. de Biologie.*) (*Israel, Virch. Arch vol. 86.*) (*Traube, Berlin Klin. Wochensch., 1871.*)

Aneurism, Varix, Stenosis.

When all coats of an artery are eccentrically dilated, aneurism is said to exist in the vessel. As a rule, one or more of these are atrophied, the inner coat the most, although the inner tunic as well as the middle may be almost obliterated, and the vascular contents held by a thickened external coat only. The aneurismal dilatations may be cylindrical in form, spindle-shaped, or as a wide irregularly-shaped sac; or one part of the widening may have one form, and another part another form. The whole length of an artery or only a small section may be thus affected. The dilatations may communicate with each other when there are several, or may remain separate.

Any of the above named alterations of the vascular wall may be the direct or indirect cause of an aneurism, yet, as a rule, diseases of the inner and middle coats, which render them inelastic and incapable of resisting the blood pressure, are liable to produce such abnormal dilatations of the vessel.

Ponfick, Virchow, Arch., 53, describes a special form which he calls *embolic aneurism,* and says that it is produced by destruction of the *intima* by calcareous masses formed upon the endocardium and carried into the circulation.

Charcot, in lecons sur les maladies, etc., holds that aneurisms formed in the brain are mostly due to lesions of the external coat and to periarteriitis.

The most frequent place of aneurism is in the *thoracic aorta* (Sec. IV, Tab. III) and in the *ascending* portion of the *arch* of that vessel (Sec. IV, Tab. II). They often attain enormous sizes, compress the adjacent tissues and cause enormous destruction of viscera by the fatal anæmia they bring about.

Next to the aorta is the popliteal artery, which is most frequently aneurismatic.

Dilatations of the pulmonary arteries are occasionally found, but they cannot be called aneurismatic, for the inner and middle coats are almost intact. (It is possible that the lesser volume and force of the right ventricular walls have not force sufficient to produce true aneurisms.) The frequency of aneurisms in the brain is very likely due to the great thinness of the arterial walls, and the very tortuous course of most of the smaller ones. In pulmonary tuberculosis, great numbers of miliary aneurisms of the finer branchlets of the pulmonary artery are very frequent.

An artery affected by aneurism never recovers, nor is its dilatation ever repaired; contrary, the vessel becomes wider and wider, until its walls are destroyed. Hæmorrhages following such destruction are more or less dangerous, according to the size and position of the vessel. Occasionally small arteries are closed up by clots in the sac, and form solid cylinders by connective tissue. The cavities of larger aneurisms are often filled, to a considerable extent, with many colored clots, arranged in strata, usually indicating the age of the

TABLE VI.

THROMBOSIS OF THE INFERIOR VENA CAVA, PHLEBECTASIS AND VARICES. PURULENT ENDO-PHLEBITIS.

CASE.—An old woman.

History.—Affected with uterine cancer. Œdema of lower extremities. Complained of much pain in the right thigh and along the femoral vein. After death the following alterations of the vessels were found:

Fig. 1. The inferior vena cava (*V.C.I.*) up to the renal veins (*V.R.*, *V.R.*) was filled with clots, adherent to the wall. A number of sacs or varices, filled with pus and other sanguinous detritus, separate from each other by thick membranes. The intima of the vessel is in many places covered with very red spots. *F.P.* is an emptied sac, showing the vascular wall. *F. P.* was not emptied, and shows its heterogeneous contents. *F. P.*, a smaller sac, with very thick walls and stratified contents, *M. M.* The two common iliac veins (*V. I. P.*) contain solid, cylindrical colorless clots. In the left hypogastric vein (*V. H.*), and a little above, to the right, newly-formed thrombi were found (*S.C.*, *S.C.*). The blood in the left external iliac vein had a peculiar yellowish, creamy color. On the right, the clot was formed at the origin of the saphena (*V.S.*), and extended into the deep and superficial femoral veins. On the left, the phlebitis extended along the whole course of the saphena vein. In the deep femoral veins colorless thrombi were found. In the collateral circulation which supplied the tissues of the lower extremities, no clots were found. The phlebitic process stopped at the newly-developed venous valves.

Figs. 2, 3 show the varices and their thrombotic contents. Also the newly developed valves where the collateral circulation begun.

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formation of the clots. Now and then from the injured walls masses of connective tissue protrude into the aneurism, but none of these collected masses ever organize, nor do they ever form a safeguard against the strong blood pressure; for very frequently the blood passes between the formed strata, softens them and causes them to be carried away into the circulation as very dangerous thrombi.

In very rare cases, calcareous masses, produced by intercurrent endoarteriitis, will close up narrow cavities of small aneurismal arteries.

According to *Charcot* (*Lecon*), cranial hæmorrhages produced in old age are always due to ruptured aneurisms of the cerebral arteries. *Eichler*, *Zenker* and *Roth* hold the same opinion.

Capillary Ectasis.

A state of capillary dilatation is always produced in chronic derangement of the circulation, when there is either stagnation or continuous turgidity of the vessels, as met with in stenosis of the mitral valves. The pulmonary and hepatic capillaries are thus found dilated. In congenital *nævus*, in muscular atrophy, in cavernous tumors, in tuberculosis, and according to *Roth*, *Hechel*, and *Virchow*, in congenital cerebral gliomata such capillary *ectasis* is found.

Varix and Phlebectasis.

Any cause producing long standing venous stagnation superinduces dilatation of a whole or a part of a vein. In the lower portions of the body, and especially in that portion of the skin where the venous circulation meets with the greatest resistance, varices and phlebectasis are most frequent. The dilatations may be cylindrical, fusiform, or like sacs. They usually become tortuous and very wide at the point of their division. When two such veins meet, they unite to form a wide sac; both cavities communicate and form a cavernous structure. Such condition is found in anal tumors (hæmorrhoids, piles). The varices often produce rupture of the vessel, with more or less severe hæmorrhages or phlegmonous ulceration, followed by metastatic abscesses. When putrid substances are there formed, or when microorganisms invade the ulcerated tissues, they become a source of dangerous and often fatal infection. Very often phleboliths, or calcareous precipitates, are formed in varicose veins. They generally close up smaller veins; larger veins they cause to ulcerate, by endophlebitis. Such ulcerations often form very infectious thrombi. At other times such thrombi organize and stop the vessels up altogether.

Solution of Continuity of Vascular Walls.

Ruptures of aneurisms and varices are the most frequent causes of spontaneous hæmorrhages. Degenerative and decaying processes going on in the vascular walls cause them to break, even without being dilated. A healthy vessel always resists the whole blood pressure, however great it may become. When an artery breaks into a tissue, the hæmorrhage from the vessel will continue until the clot formed is capable of resisting the blood pressure. Such clots are called *hæmatomata*. The rent in the blood vessel is closed up by colorless corpuscles and *blood discs*, which gradually form a colorless mass extending into the vessel, and protruding outside. (*Schultz.*) The internal portion of the mass becomes absorbed, whilst the external remains continuous with the external wall. Adhesive inflammation always following such ruptures, gradually forms a sac around the external clots. Such a sac is called a false aneurism. This kind of aneurism often breaks through and causes great hæmorrhage, or is filled up by newly-formed connective tissue within the sac.—(*Schultz.*) Vessels derived from the repaired vascular wall, often start cicatricial tissue and extend it over the sac, and eventually obliterate it by cicatricial constriction. Venous wounds are often healed in this manner. Such sacs are called false varices.—(*Schultz.*) Gun-shot wounds of blood vessels, are, very often, spontaneously repaired in this manner.—(*Schultz and Klebs.*)

Aneurisma dissecans. Aneurismal Varix.

When it happens that only the inner and middle arterial tunics are broken, but not the external, the blood does not enter the tissue, but passes between the coats. The external coat becomes tumified, and the greatest portion of it is severed from the other coats. Such a condition, which is frequent in the ascending aorta, is called *aneurisma dissecans*. The clots are sometimes so extensive as to loosen the whole extent of the external vascular coat from the others, and the separation only ceases at the entrance of the vessels into the parenchymatous organs. The quantity of the blood between the tunics is occasionally very great. (In Sec. IV, Table IV the aorta is thus dissected to a great extent.)

When an aneurismal sac of an artery is united with a vein by adhesive inflammation, the latter becomes compressed, the walls are gradually destroyed, and the aneurism breaks into the vein. It forms an aneurismal varix. When a vein and an artery are together perforated, for instance, by a *stab*, a false aneurismal varix will ensue. When an opened artery breaks into a vein and dilates it, then an aneurismal varix is formed. The vein becomes dilated and thickened.

Thrombosis. Intravascular Coagulation.

Death of the blood is generally followed by coagulation, that is, solid masses are separated from the liquid portion of the blood, as so called *fibrin*.

Coagulation taking place in a vessel during life, is called thrombosis, the coagula, thrombi.

When the blood is stagnant in the vessel, the thrombus is of a dark red color, and consists of granular albuminous masses, a few colorless, and a great quantity of colored corpuscles.

Vessels closed by permanent stasis contain red thrombi, which are at first soft and spongy, containing blood serum. They grow more solid, contract and become reddish-brown. In still flowing blood only portions of it are precipitated as coagula, which are made up chiefly of colorless corpuscles and blood discs (*Zahn*, *Bizzozero*); they are grayish or reddish yellow, or mixed in lighter and darker colored layers. White thrombi often collect along the vascular wall, and contain great quantities of fibrin.

As the endothelium of the vessel is capable of keeping the blood in a liquid condition, whenever this tissue becomes diseased, or is unable to perform its function, the blood will coagulate, the same as when it dies from poison or becomes stagnant from mechanical causes. Endocarditis as well as endoarteriitis are the most frequent causes of thrombosis. Thrombi may be formed along the vascular wall, along the venous or cardiac valves. They may close the vessel up either by primary or secondary formations, or may at first occlude it and then by contraction of the thrombus free a portion of the cavity and render circulation possible through it. Thrombi are always formed in such localities and under such circumstances, where and when the blood is deprived of its contact with the endothelium and is stagnant. They are therefore more frequent in veins, at the valves than any where else, and spread from the smaller into larger veins.

Parenchymatous inflammations resulting in gangrene are the most frequent causes of thrombosis of small vessels.

The most favorable results are those in which the perfectly formed thrombus becomes contracted and hardened, by absorption of its liquid portions. They are then as a rule converted into calcareous masses, and either close up the vascular cavity by causing their walls to shrink, or leave a portion of the cavity open to re-established circulation. *Phleboliths* are calcareous masses formed in the centers of thrombi. As a rule, new endothelium covers such cretaceous masses and prevents the formation of new thrombi.

Softening of the thrombotic centra are far less favorable to the vessels and the tissues enclosing them. The softened portions either form red or grayish-red masses of corpuscular and granular debris and blood crystals, which, when carried into the general circulation, form dangerous emboli.

Purulent and gangrenous, or yellow and greenish softened masses formed in the centers of the thrombi, consist of decomposed and decayed fibrin, blood corpuscles and other detritus of the blood. They act not only as inflammatory foci upon the adjacent vascular walls, which they corrode and cause to undergo purulent inflammation, but also the gangrenous particles, usually found to contain highly putrid substances and large quantities of micrococci, completely destroy the inner and middle coat, and both become the most dangerous sources of putrid infection to the whole circulation.

Purulent thrombophlebitis is called the purulent softening of a venous thrombus with infiltration of its walls.

Organization of the Thrombus.

The new tissue is formed from infiltrated colorless corpuscles. The endothelium of the vessel has generally but little to do with the tissue reparation, whilst the thrombus itself acts more as a slight irritant upon the newly developed connective tissue. Newly formed vessels gradually penetrate the organizing mass of spindle shaped cells, and form a membrane very much like a newly forming serous membrane. The intervascular spaces gradually fill up by infiltration of round cells, which develop into connective tissue cells. The tissue formation in such organization differs in young and vigorous individuals and old or anæmic persons. Emigration of round cells into the organizing thrombus takes place from the vasa vasorum of the thrombotic vessel itself and the nearest capillaries.

The healing of ligated vessels is thus brought about: A thrombus is formed above the ligature, consisting chiefly of colorless

Fig. 1

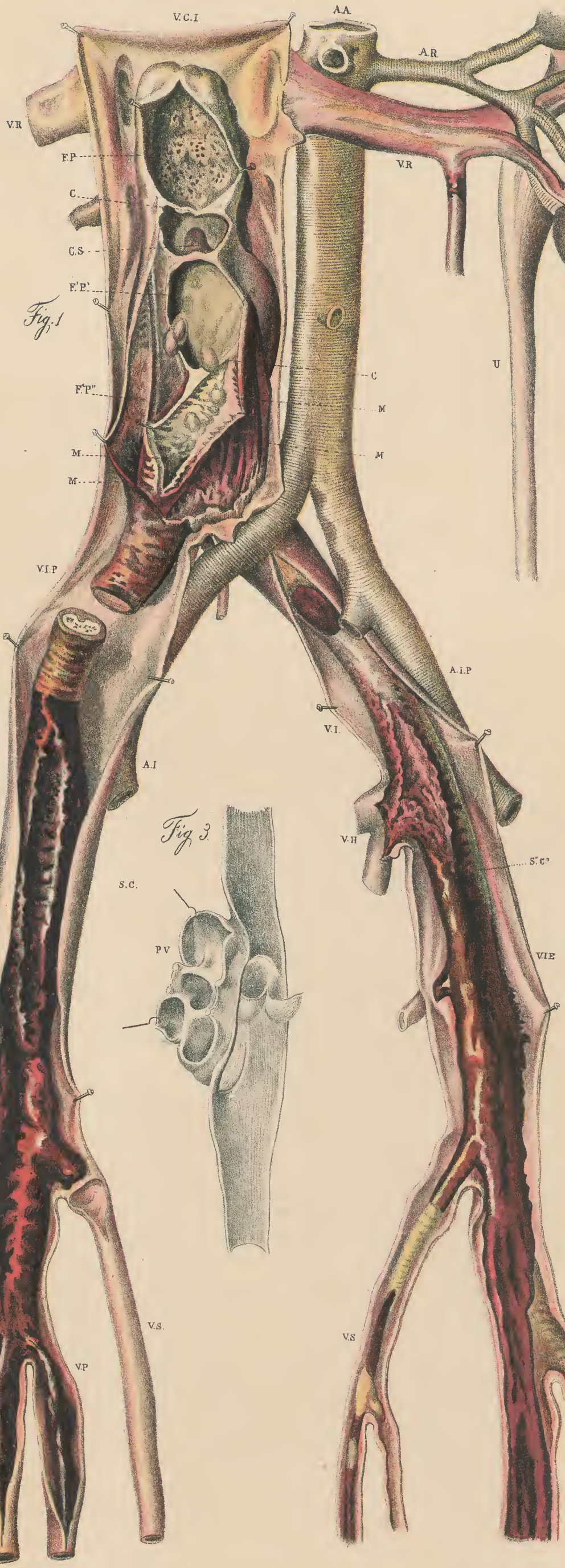
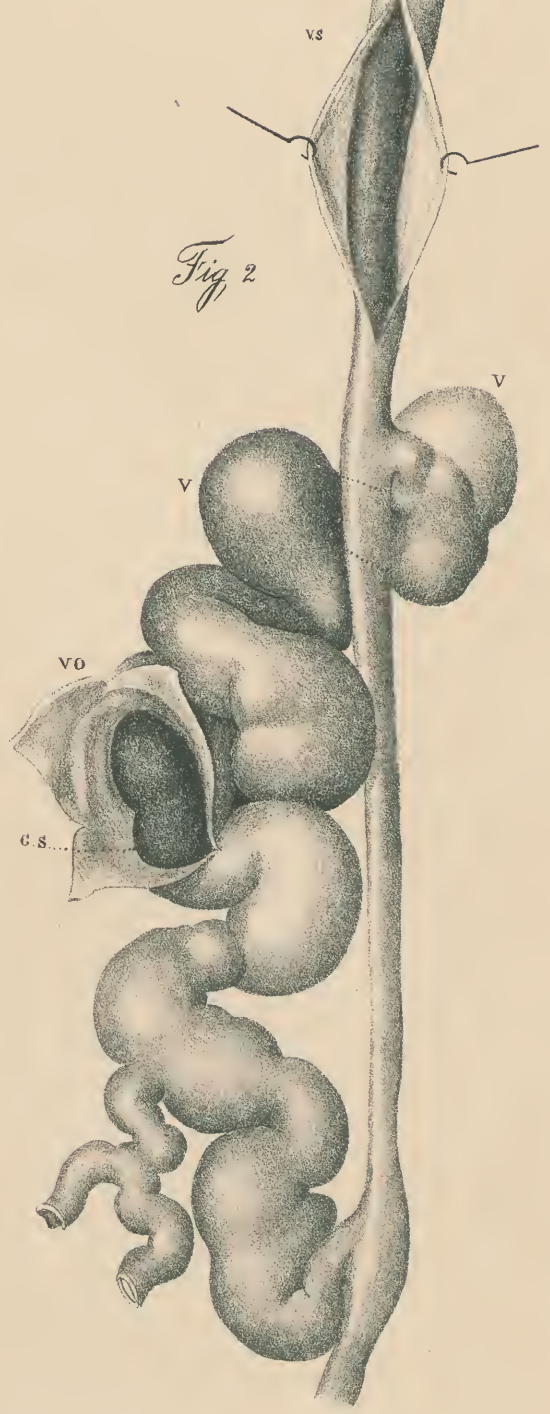


Fig. 3

Fig. 2



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Sec. IV. *Tab. VII.*

ENDO-PHLEBITIS OF SUPERFICIAL VEINS OF THE ARM. INFECTIOUS THROMBI.

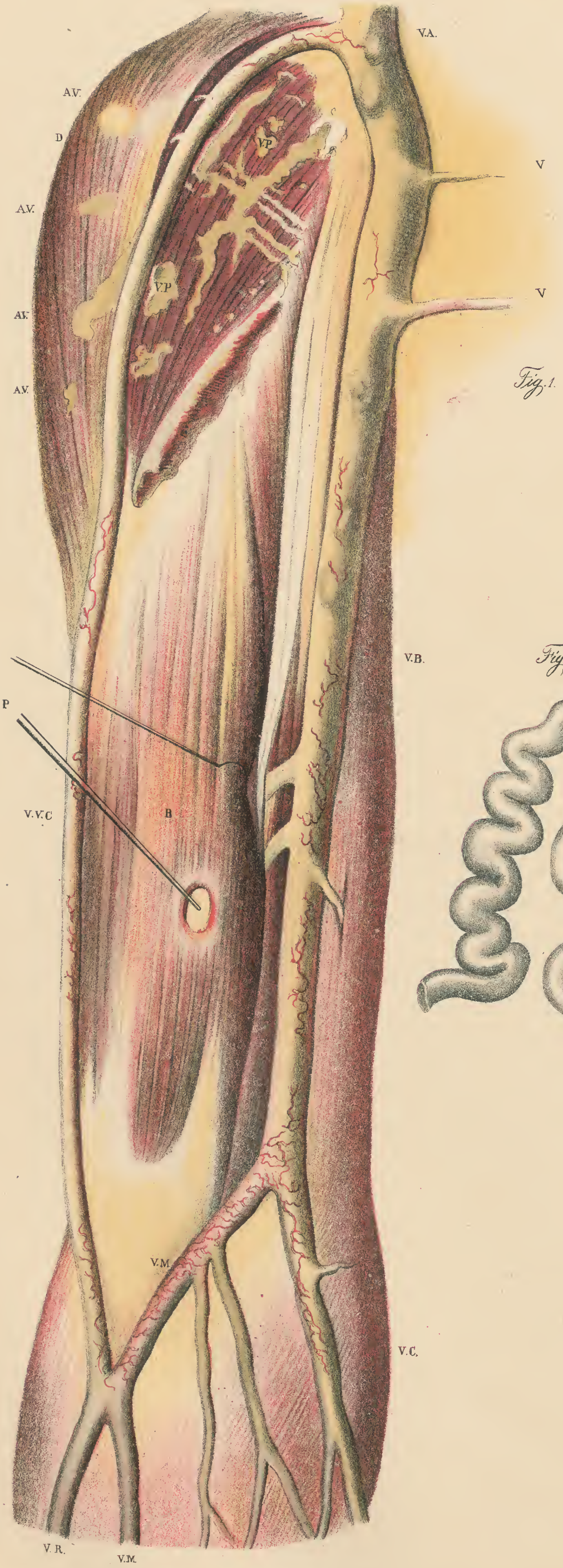

Fig. 1.

Fig. 3

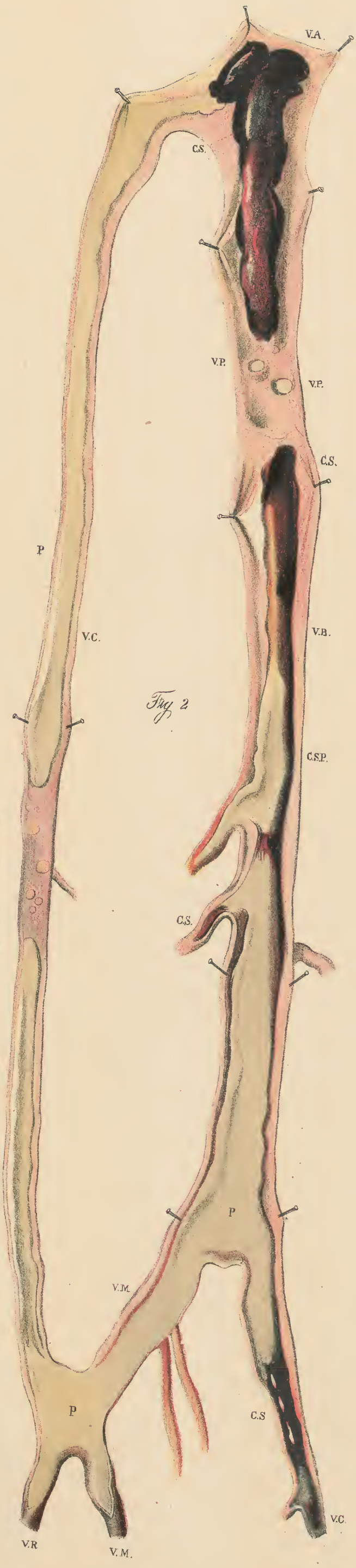

Fig. 2

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TABLE VII.

ENDOPHLEBITIS OF THE RIGHT MEDIAN VEIN AND ITS BRANCHES FROM INFECTIOUS THROMBI.

CASE *B.*—*Xavier*—Young man, twenty-five years old.

History.—Was accidentally shot in the arm. The weapon was loaded with small buckshot, one of which passed through the lower third of the limb; the other slightly deviated from its course (antero-posterior) and was lodged under the skin, on the internal surface. The shot was readily extracted and the necessary care given to the patient. The humerus, the principal vessels and nerves of the arm, seemed not at all hurt; everything seemed very favorable, yet on the twenty-fifth day after the wounding, the limb began to swell and suppuration suddenly ceased. Local and general symptoms of phlebitis appeared; visceral inflammation spread rapidly; the patient became pyæmic, and after six days died of acute septicæmia.

Post mortem.—Endophlebitis of the larger and smaller veins of the arm; multiple metastatic abscesses in the deltoid and in the muscles situated anteriorly on the forearm; bilateral lobular pneumonia; right-sided pleuritis; granular hepatitis.

Fig. 1 shows the biceps perforated by two shots. The cephalic (*V.C.*), basilic (*V.B.*), ulnar, median and radial veins (*V.C.*, *V.M.*, *V.R.*), and all their branches, are cylindrical, tense, very voluminous, and, in places, very much tumefied. The connective tissue is exceedingly infiltrated, the vasa vasorum injected and plainly visible.

Fig. 2 shows the opened veins, their walls are thickened; the cephalic vein is full of pus, likewise the median and radial veins (*V.H.*, *V.R.*); the basilic presents all stages of phlebitis; a great many purulent cavities were found within the thickness of the deltoid (*A.P.*). It was very plain that these abscesses were produced by multiple endophlebitis of the smaller veins. The other muscles besides those described were perfectly sound. The scapulo-humoral cavity was entirely filled with pus.

Fig 3 shows the tortuous course of chronic venous thrombosis, often produced by stagnation of venous circulation.

blood corpuscles. Shortly afterward the vessel becomes slightly tumefied, and is filled up by new blood corpuscles, which develop into connective tissue cells, and with the reformation of vascular net-work, close up the wound produced by the ligature.

When harmless thrombi find their way into the general circulation, they generally locate in small vessels, or at the origin of vascular branches. Upon such emboli, as the stagnant masses are now called, new coagula are deposited, which may completely close the vessel up; or they may undergo the same changes as the primary thrombi, etc., as described above. (*Schultz Deutsch Zeitsch. f. chirurg, IX Vol.*) (*Raab, Arch. f. Klin. chirurg, XXIII Vol., Virch. Arch. LXXV.*) *Riedel, Deutsch. Zeit. s. f. chirurg, 1875.*) (*Baumgarten, Sogenante Organizat. des Thrombus, Leipzig, 1877.*) (*Durante, Wiener Med. Jahr. III, IV.*) (*Senftleben, Virch. Arch., Vol. 77.*) (*Tillmann, Virch. Arch., Vol. 78.*) (*Auerbach, Diss. Bonn, 1877.*)

PHYSICAL DIAGNOSIS OF ARTERIES.

By means of inspection, auscultation, percussion and sphygmography, disturbances of arterial functions and their lesions can be readily ascertained.

Pulsation is the visible manifestation of activity in arteries, in the shape of a rhythmical wave movement, more or less synchronous with the systole of the ventricles of the heart.

In perfect health, and perfectly at rest, even the larger arteries do not show distinctly visible pulses. But when the activity of the heart is in any way increased, or its movement in any way accelerated, a lively beating in the lateral aspects of the neck, and a rhythmical vibration in the jugular fossæ are plainly visible. Even in such small arteries as the temporal, pulsation then becomes distinctly visible.

Capillary pulsation has been noticed by *Quinke* and *Lebert* in insufficiency of the aortic valves, in aneurisms and paralysis of the middle coat of the larger arteries. Like the abnormal activity of the heart, which indicates either its own functional disturbances, or anatomical lesions, the arteries, by their abnormal activity, will show either functional derangements of the circulation, or structural lesions of their own tissues. Differential diagnosis of local and general disturbance of the circulation, by the changed pulse phenomena, can only be made by the aid of percussion, auscultation, etc., of one or more parts of the body.

A peculiar pulsatory movement is often visible in the epigastrium, constituting a rhythmical systolic vibration of the abdominal walls, and occupying the space between the xiphoid cartilage and the adjacent costal cartilages. The vibration sometimes extends to the umbilical region, and even beyond it. This vibration is produced by the heart directly, or by the larger arteries.

Only when the heart occupies a very low position, and the diaphragm is very much depressed, can the epigastric pulsation be ascribed to direct action of the heart. In this case, the apex beat will be found very low in the thorax, or, not at all, but auscultation will demonstrate the presence of the cardiac pulse in the upper epigastric region, and occasionally the ventricular systole may be felt, when the abdominal walls are very thin.—(*Eichorst.*)

Epigastric pulsation produced by the aorta, by distention of the stomach or other intestines, carcinoma of the œsophagus, cerebro-spinal meningitis, by a number of neuroses, produced by direct irritation upon the peripheral nerves, or by reflex action upon the vasa motor centra, will be indicated (besides other coordinate symptoms) by the pulsation taking place after the cardiac systole.

The same is the case in extensive aneurismata of the abdominal aorta. Aneurismal enlargements in superficial vessels present themselves to the eye as pulsating tumors, whereas in deep arteries they do not become perceptible as a pulsating tumor until the overlying tissues have become absorbed or have been displaced by them. Differential diagnosis between a solid tumor lying against a large artery, and moving with the pulsating vessel, and an aneurism of an artery can only be properly established by palpation, In case of a solid tumor, only the artery will be felt to really pulsate, whilst in an aneurism there will be *enlargement* of the sac after each systole of the heart.

Very peculiar changes take place in the arteries when the aorta is either constricted or totally occluded at the opening of the Ductus Botalli; for under such circumstances only collateral circulation can supply the lower half of the body with blood. The collateral vessels increase enormously in size, and become perceptible as large pulsating cords. There are three ways in which collateral circulation can be established in such cases:

1. By the subclavian, the internal mammary, superior epigastric, inferior epigastric and external iliac arteries.
2. By the subclavian, internal mammary, anterior intercostal, posterior intercostal arteries and the descending aorta.
3. Subclavian, transversalis colli, dorsalis scapulæ, posterior intercostal arteries, and the descending aorta.

Palpation of Arteries.

The stronger the pulsation the more palpable the artery to the touch. In aortic insufficiency a short fremitus is perceptible in the carotid and subclavian arteries. In very thin people such a fremitus may artificially be produced by pressure with the finger or with the stethoscope upon the larger arteries or the abdominal aorta. It differs from a fremitus morbidly produced, by the absence of murmur, which is nearly always present in aortic insufficiency.

Auscultation of Arteries.

In order to auscultate arteries, even the larger ones, great care must be had to keep the person very quiet, for the slightest displacement will very often change the tension of the vessel, and with it the sound. An arterial sound is normally short, quick, and circumscribed to the locality. Presence of murmur, or *bruits*, denote disease. These may originate in the heart or in the arteries. Mechanical obstructions will sometimes give rise to murmur, without the vessel being affected; for instance, when the elbow is bent, pressure upon the brachial artery will yield a murmur which grows in intensity up to a certain point, which will again decrease and disappear when the pressure becomes very great. But when the pressure is at the highest, there will be a quick, sharp sound, but no murmur. Completely closed vessels yield no sound whatever. The subclavian and the carotid arteries in the healthy emit a very distinct sound. In the majority of cases, the common carotid, at its origin, has two sounds, synchronous with the filling, and subsequent contraction of the artery. If only one is present, it is always during arterial systole, and will then indicate weakness of the vascular wall. In the subclavain artery the same phenomena takes place. In all large vessels sounds may originate in consequence of local changes of its contents or its walls. A sudden contraction or dilatation of an artery usually produces a tone. Murmurs in the pulmonary artery denote constriction or compression of its walls. Chronic inflammation or thickening of the left lung, or tumors which compress that vessel, produce arterial sound.

Aufrecht describes a case in which he heard a systolic and diastolic murmur in the region of the pulmonary artery, which was most intense in the third left intercostal space, about three centimeters from the left edge of the sternum. Post mortem examination showed that the left lung contained no air at all, but was filled with cartilaginous detritus. The main branch of the left bronchial artery was larger than the trunk at its origin. The other branches of the bronchial artery within the lung were exceedingly reduced in size. The change in the caliber of that vessel caused the sound.

Litten describes a case of systolic murmur, over the region of the pulmonary artery, caused by an embolus in a large branch of the bronchial artery. Murmur in the subclavian artery may be caused by pressure produced upon it, by the scaleni, the subclavian muscles, or by the clavicle, against the first rib. In consumptive patients subclavian murmur is a very frequent phenomenon, for, usually in these cases, the arterial coats and the pleura are united by adhesion, and the vessel becomes alternately stretched and twisted by breathing, thus continually changing its caliber. These murmurs are either unilateral or bilateral.

An intermittent, blowing sound, synchronous with the carotid pulse, is occasionally heard over the cranial surface of children, from the third month up to the sixth year. It is loudest over the posterior fontanelle, and reaches sometimes the region of the first cervical vertebra. It is attributed by some authors to the longitudinal sinus, by others it is located in the basal arteries. *Jouarez*, finding that the blowing is synchronous with the carotid sounds, considers it a continuation of these. As the carotid canal is

TABLE VIII.

MULTIPLE CAVERNOUS ANGIOMATA. SENILE GANGRENE.

Figs. 1, 2, the dorsal surface of the right hand of an individual affected with cavernous tumors over a great part of the body. Those on the body resembled fibroma molluscum; on the hand their cavernous character was distinctly marked. The tumors on the hand, Fig. 1, are small and spheroid; the fingers are extremely deformed. Fig. 2 shows the palmar surface of the same hand, the skin dissected away; the tumors are seen to be occupying every tissue of the member. Before removal of the skin, they looked bluish and purplish, nearly spherical; the skin over them was very fine, but healthy, and could freely be moved over them, backward and forward; some looked like ripe mulberries; after the skin was removed, the tumors presented a racemose appearance, were freely movable, but held to their places by a very fine connective tissue. They all constituted an erectile, cavernous or spongy tissue, in different stages of morbid alteration.

In some of the tumors the blood was liquid, and appeared to have had free circulation; some contained coagula of different colors; some contained small phleboliths of different sizes, in the thrombi; the larger phleboliths were surrounded by thin clots, which were mostly colorless; these calcareous secretions were of different degrees of hardness.

This person was affected with a weak heart, and general scrofulous habit. (*I. V.*, vascular tumors.) (*P. L.*, multiple tumors.)

Figs. 3, 4 show the dorsal and plantar surfaces of the foot of a woman of eighty-five years, affected for years with excessive varicosities on both legs. The gangrene, which developed on the right foot, began at the little toe, and slowly spread upward. She died from pleuro-pneumonia and pachymeningitis. Most of the arteries of the body were found atheromatous.

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constantly undergoing a change of growth, etc., during infancy, there is great plausibility for the theory, that the murmur is due to change of caliber of the arteries, especially as in the middle meningeal and in the temporal murmurs are often enough found to exist at that period of infancy. In aneurism, the sounds and murmurs are so constant in different portions of the enlargements and constrictions, and synchronous with the diastole of the artery, that no other cause can be ascribed to them, than the eddies, formed in the sac. Aneurismal *bruits* are often exceedingly loud, and audible at a distance. When the interior of the tumor is so filled with clots as to absorb all sound and to leave but a very narrow passage for the blood, no bruit or murmur will be heard. In extensive dilatations and tortuousness of arteries, as is often found in such diseases as that of Basedow, etc., murmurs will be found over the region of the crooked vessels. Congenital stenoses of the aorta, near the origin of the ductus, Botalli is liable to produce fluctuation in the arteries, and render them tortuous. Fremitus, murmurs, etc., may be looked for in several regions of the body in such cases. Leopold lately discovered an arterial murmur in a case of cancer of the liver. There was also a constant sound, which became louder during every arterial diastole. He ascribed them to excessive dilatation of the hepatic vessels. In excessive inflammations of the uterus and ovaries, murmurs and sounds are very frequent, and are owing to the existence of dilatations and contractions of the vessels of those organs. When two opposite blood currents meet, for instance, when an artery breaks into a vein, eddies are produced, and with them there will always be murmurs of greater or lesser intensity. Cossy cites a case of rupture of an aortic aneurism into the vena cava superior, producing fremitus and *bruits* in the jugulars.

In general anæmic and cachectic condition of the body, arterial *bruits* and murmurs are a very common occurrences.

DIAGNOSIS OF DISEASES OF THE VEINS.

Inspection of the veins must be carried on upon the larger superficial veins of the body, to ascertain their characteristic state of tension, fullness, and the mode of movement of the blood in their cavities, especially during respiration.

Venous plethora may be local or general, and in either case a greater or lesser obstacle to circulation will exist. Thrombi in the veins, or tumors adjacent to them, usually form such obstacles. The distal end of an occluded vein is generally found distended and very tortuous in its own course (Sec. IV, Tab. III) as well as in that of its branches. Diseases of the portal circulation, as well as in the liver, very frequently produce such phenomena in the veins of the abdominal wall. In pregnancy, or when there is much pressure upon the vena cava, similar venous conditions are found in the lower extremities and in the abdomen. Mediastinal tumors and aneurismata of the abdominal aorta likewise produce such effects upon the abdominal walls. Dilatations and thrombosis of the transverse and superior petrosal sinuses dilate the internal jugular, while the external jugular becomes much reduced in caliber.—(*Gerhardt.*)

Diseases of the lungs, the heart, and abdominal viscera produce enormous modifications in the tension and caliber of all veins, very often visible in the surface veins. Venous stasis is most perceptible in the veins of the neck, especially when the patient occupies a supine position. The external jugular may assume an extraordinary width, and the internal may even become wider than the external, and appear from above downward, as a large tumor, behind the sternocleido-mastoid muscle, and its sterno clavicular attachment, when the person is coughing, or when any pressure is brought to bear upon it.

Visible respiratory movements of the veins.

Inspiration assists the flow of venous blood toward the thorax, expiration, to some extent, checks it. In healthy persons such respiratory movements of the veins are but very seldom perceptible. But when veins are overfilled, from any cause whatever, or when respiration goes on irregularly, then each inspiration will decrease, each expiration increase the volume of the veins of the neck.

Pulsation of veins is usually connected with venous plethora. Such pulsation must be differentiated from simple undulation, produced by an overlying vein upon an artery. When there is a modified functional or anatomical state of the right heart, venous pulsation can be looked for, especially when the heart-force is much reduced. Normally there is a slight retrogression of the venous blood into the venæ cavæ during the auricular systoles; but when from any cause there is disturbance in the pulmonic, or in the coronary circulation, etc., then the backward wave becomes visible. The external jugular becomes very prominent during the presystolic phase of the heart's action. Occasionally a double venous pulse is found, one presystolic and the other simultaneous with the ventricular systole, but ordinarily the internal jugular, and afterward the external, pulsate. Gerhardt and Seidel found, under the above described circumstances, pulsation of many cutaneous veins. Walsh found a pulsating mammary vein; Raridan, in a case of cirrhosis of the liver, a pulsating epigastric vein; Geigel a pulsating inferior vena cava; Seidel and Marey, pulsating venæ saphenæ. Pulsation of the internal jugular will denote insufficiency of its valves, and may be produced very suddenly. Such insufficiency may be caused by insufficiency of the tricuspid valves, when there is at the time hypertrophy of the right heart. Reisch and Rosenbaum found such venous insufficiency in incompetency of the mitral valve and lack of closure of the foramen ovale. Stokes and Friedreich met the same in exudative pericarditis. It is always presystolic, and as a rule cannot be recognized by the sphygmograph. Seidel's researches have shown the great symptomatic importance of pulsating hepatic veins. It is hardly ever present except in insufficiency of the tricuspid valves, and is really more valuable as a symptom than jugular vein pulsation, for it appears long before the neck veins commence to pulsate, since the inferior vena cava has no valves, and any regurgitation of the auricle toward the venous system will necessarily affect this vessel first. This pulsation is most perceptible over the right lobe of the liver. The cause of the hepatic vein pulse was thought to be the raising up of the liver by the over-filled vena cava; but, firstof all, the organ is too voluminous to be lifted by that vessel, and secondly, as Taylor and Thamm have shown that the pulsation is to be felt as a vibration below that gland (*the hepatic vessels*), it can only be ascribed to a turgidity of the hepatic veins, caused by the backward wave in the inferior vena cava. The pulse of the hepatic vein, sometimes, suddenly disappears to reappear. Weakness of the heart renders it imperceptible. Aneurismal enlargements of the abdominal aorta, or, especially, of the hepatic artery, often cause the liver to pulsate, or rather undulate; it differs from the hepatic vein pulse in not causing any temporary enlargement of volume of the liver, which finds place in the venous pulsation.

Pulsation in the inferior vena cava and in the veins of the lower extremities are of very rare occurrence.

Auscultation of veins.

Sounds, or tones and murmurs, are audible in the veins. When the blood current becomes suddenly widened or narrowed, murmurs arise. Venous sounds are usually characterized by not being ryhthmical like the arterial. Now and then a rhythmical venous murmur is to be heard, then it is generally due to some pressure brought to bear upon the vessel.

As a rule, venous sounds are due to vibration of their valves by the retrograde wave of the blood. It must be stronger where the force acting upon the valvular membrane is strongest, and necessarily absent where there are no valves. Yet a number of very conscientious observers have perceived tones and murmurs in valveless veins. Such sounds must, of course—if they really do exist—be ascribed to the blood current acting directly upon the vascular wall.

Normally a murmur is heard in the bulb of the jugular vein. Laenec, who first described it, thought it was due to a continuation of sound in the carotids. It is characterized by *a low, continuous hum*, which varies in intensity every few minutes, so as to produce quite a scale of murmurs. When it is very intense, it will annoy the person as a disagreeable, buzzing sound in the ear. It is usually ascribed to increased velocity of the blood toward the heart, as it is mostly audible when the person stands upright, or by deep inspiration, whilst by deep expiration, and in recumbent posture it becomes very, weak or disappears altogether. It has nothing to do with a pathological backward wave, and is synchronous with cardiac diastole. This murmur used to be designated as *bruit de Diable, Bruit de Nonnes*, and was considered a valuable symptom of chlorotic and kindred diseases, but is really of no value whatever, as it is a very normal phenomenon, and simply indicates the frictional condition of the blood, according as it moves faster or slower toward the heart. Weil and Friedreich have found similar murmurs in the subclavian and other large veins, but which were all devoid of clinical interest.

CANCRENA SENILIS.

MULTIPLE CAVERNOUS ANGIOMATA.

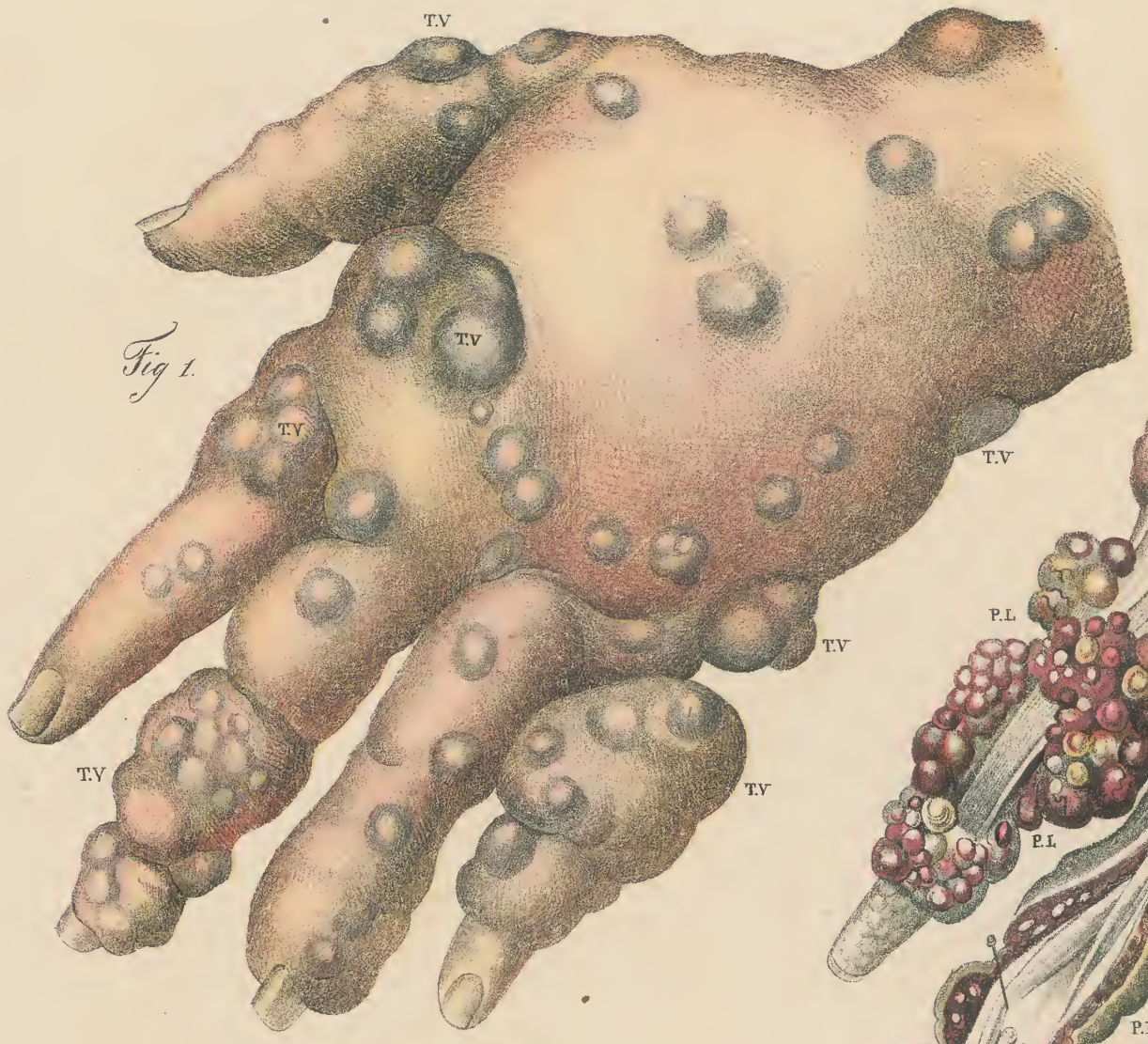


Fig 1.

Fig. 2.

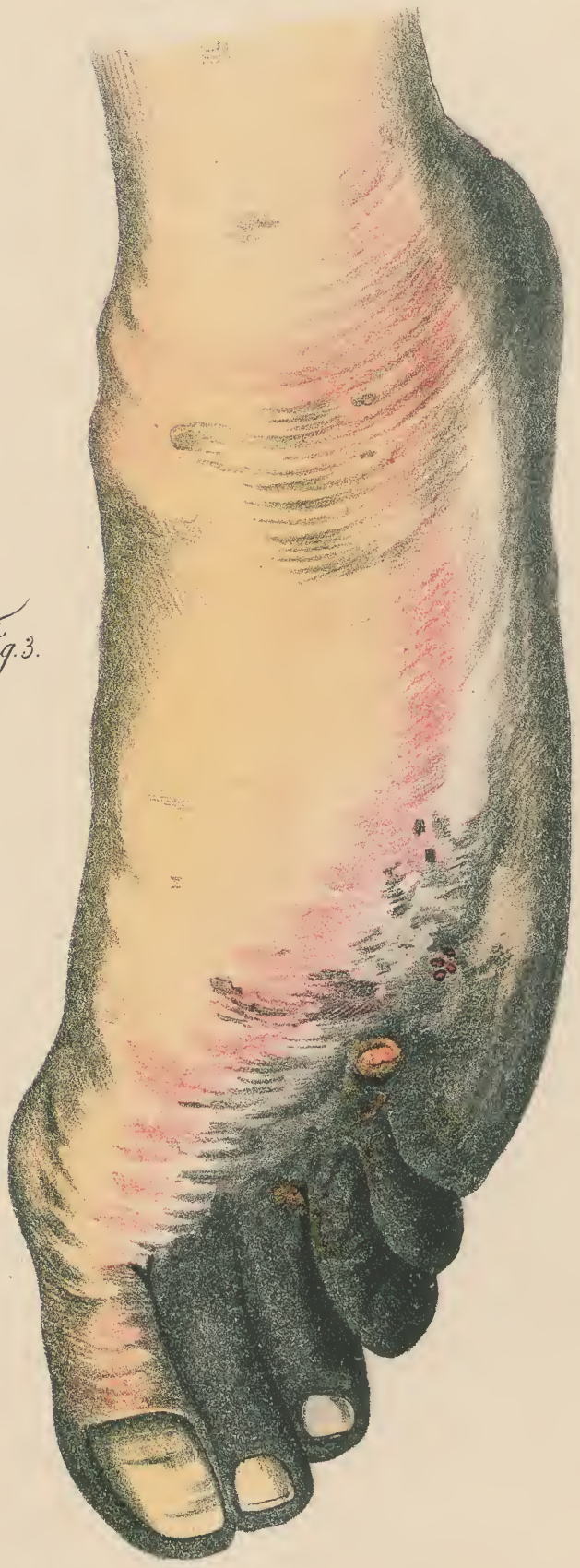
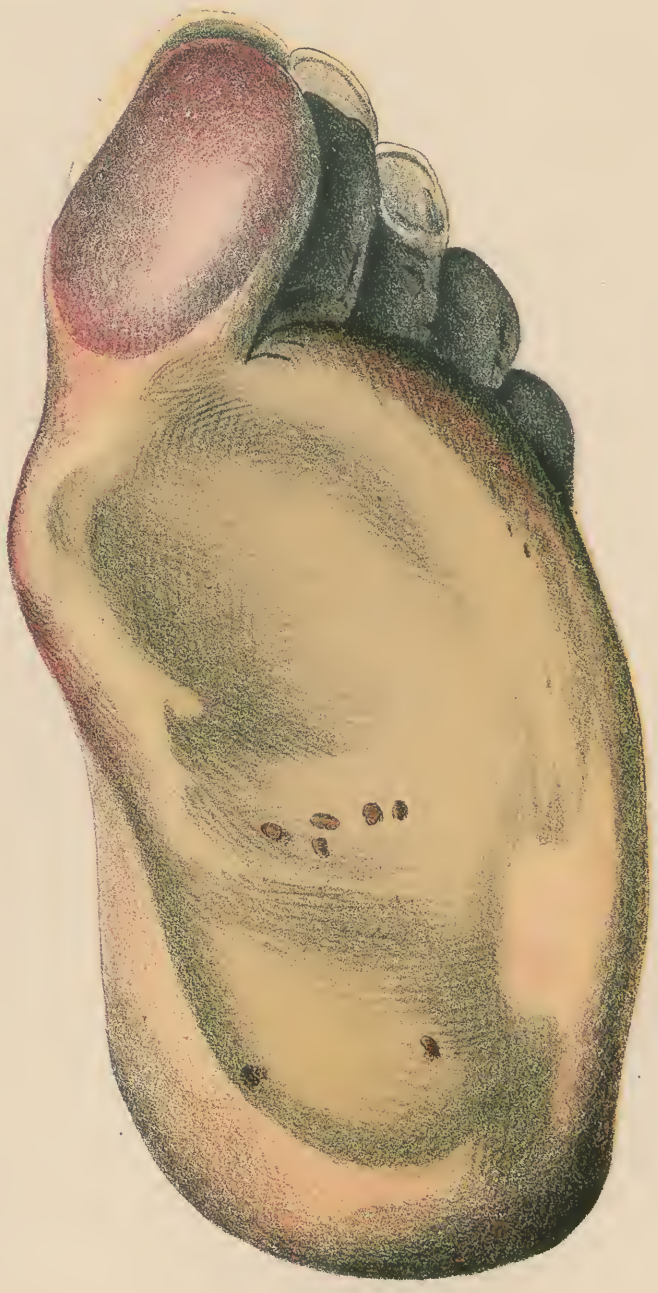


Fig. 3.

Fig. 4.



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DISEASES OF SPINAL CENTERS OF PERIPHERAL NERVES.

CARIES OF DORSAL VERTEBRAE

SPINAL PACHYMENINGITIS.

Sec. V. *Tab. I.*

Fig. 1.

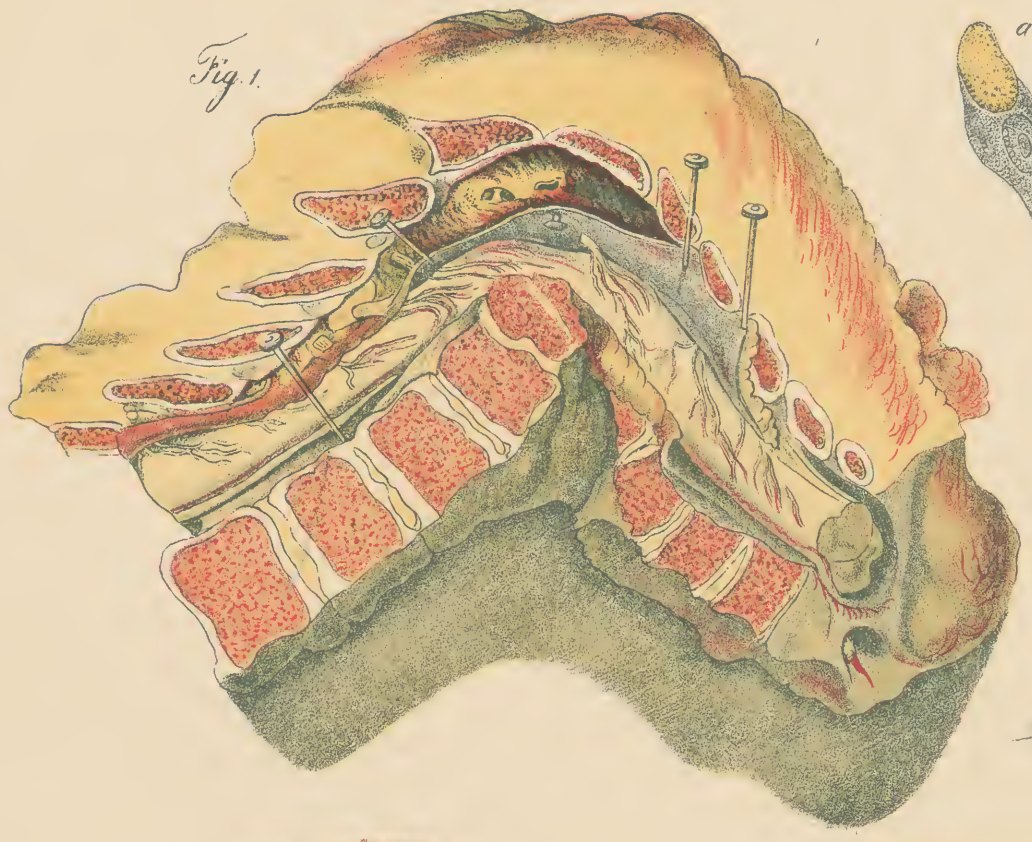


Fig. 4

Fig. 2.

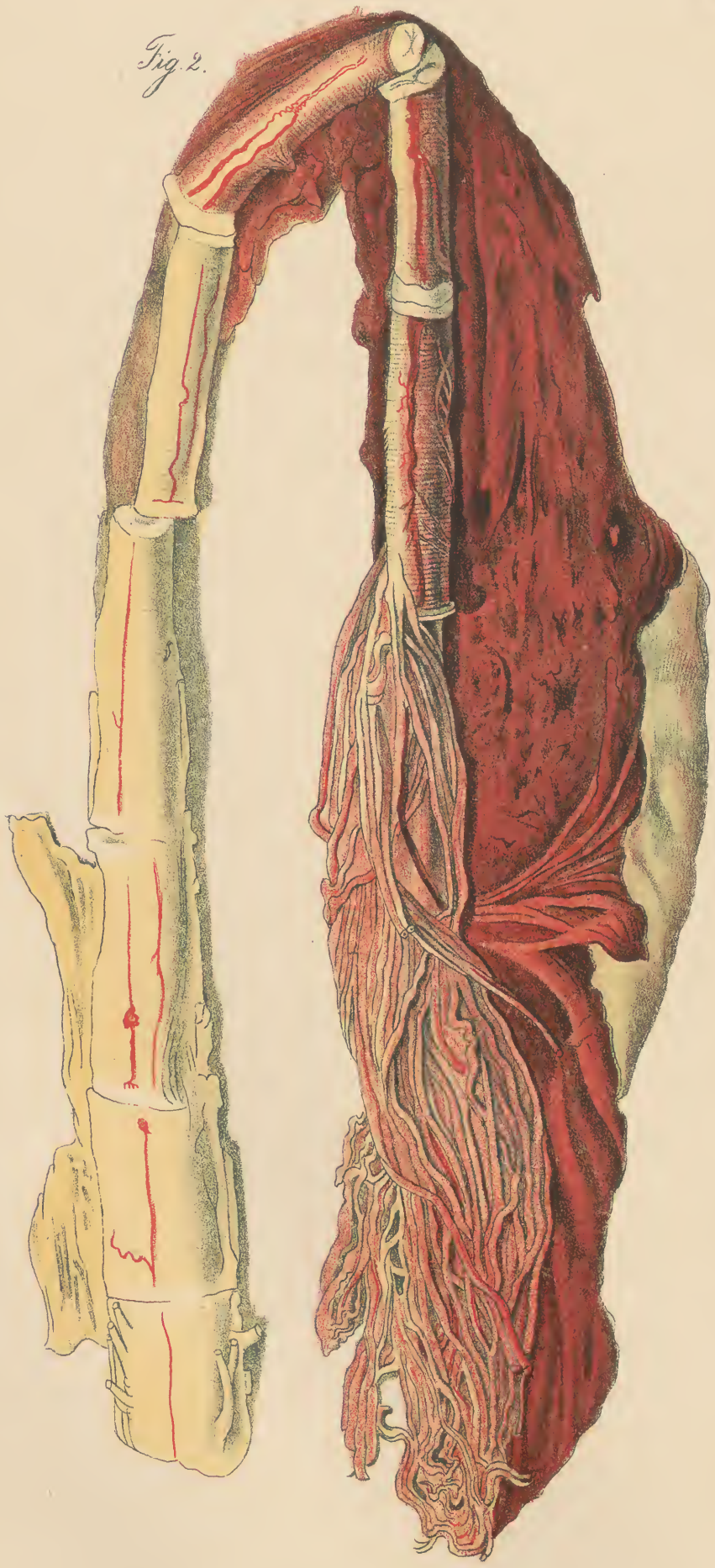


Fig. 3.

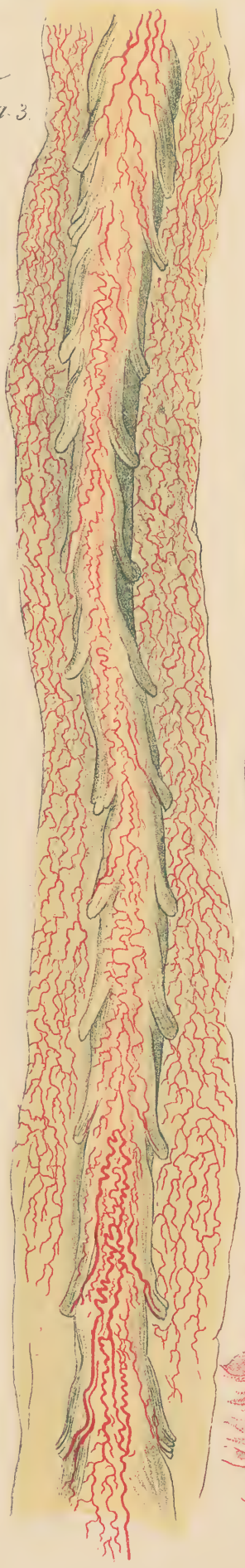


Fig. 5.

Fig. 6

Fig. 7.

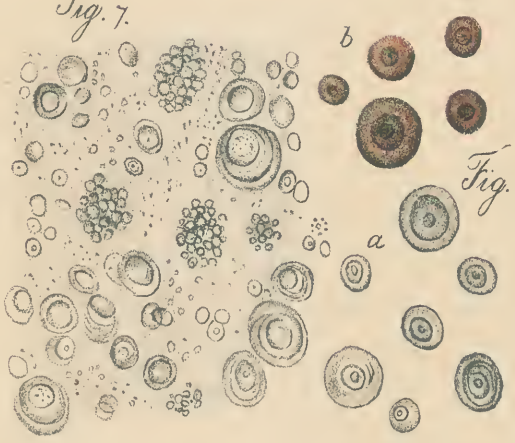


Fig. 8.

Fig. 9.

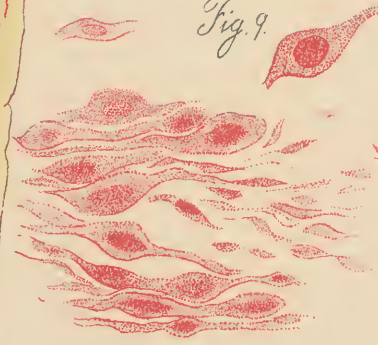


Fig. 10.

TABLE I.

FIG. 1.—*Caries of Middle Dorsal Vertebræ, with Posterior Kyphosis.* The spinal marrow in the curve is much compressed, soft, and of a grayish-red color. The cellular structures behind the *dura mater* are infiltrated with pus, and there are many ecchymoses of a deep red color.

FIG. 2.—*Spinal Pachy-meningitis.* The affected person was weakly and suffered for many years with rheumatism. The attack of meningitis was sudden, after a fall upon the head. Was at first senseless, but soon recovered; became paretic, and had to be in bed. Ten days after the attack his speech became indistinct, and he fell into a semi-comatose state, with involuntary discharge of urine and stools. Temperature and pulse nearly normal; respiration slow and sometimes irregular. Both pupils alike contracted. Stinging the face with a needle causes contraction of its muscles. There is partial muscular paralysis of the upper and lower limbs. Sensation but little disturbed. Considerable stiffness of neck and back. There is no deformity nor painful places on the spine; abdomen flat and not tender. After improving for several weeks he had a relapse, became insensible, coughed, and expectorated very copious, highly offensive sputa. With sudden rise of temperature and very frequent pulse he expired a month after the accident. Autopsy (by *Prof. Recklinghausen*): Gangrenous bed sores on sacrum. The spinal dura mater very much dilated, flabby, and filled with liquid. In the lumbar region great plaques filled with calcareous matter were spread over the outer surface of this membrane; its inner surface was covered with red, rust-colored, pseudo-membraneous exudate, in which were disseminated very many punctiform hæmorrhages. The origin of the filum terminale was also of that rust color. The pia mater was lightly yellow in spots; in some portions ossoid plates. On the posterior surface the vessels were much injected. The spinal marrow was slightly softened in its whole length, containing many hæmorrhagic spots, but otherwise unaltered. At the base of the brain the dura mater was of a brown color, and covered with similarly-colored exudate; the pia mater a little yellow. There were several sphacelus and gangrenous foci in the lungs. There was a considerable fissure in the temporo-parietal region of the skull, where the inflammation very likely originated, from the fall, and from there extended downward into the spinal cavity (*Leyden, Klinik d. Rueckenmarks Krankheiten*, page 406).

FIG. 3.—Tubercular spinal meningitis. The mia mater on the posterior aspect of the spinal marrow is much thickened, and the injected vessels are but slightly perceptible. The inner surface of the dura mater is opaque, and streaked in different directions; it is covered with numerous gray or yellowish-brown tubercular nodules.

FIGS. 4, 5, 6, 7, 8, 9, 10.—Morbid Histology.

FIG. 4.—Morbidly-altered ganglionic cells. (A a a), pigmented cells in old age; (b b b′ b′), enlarged ganglionic cells in traumatic myelitis; (c c c c), atrophied cells in progressive paralytic dementia; (d d d), atrophied cells in progressive muscular atrophy; (e), cells from a degenerated anterior cornu.

FIG. 5.—Fatty degeneration and atrophy of the root of the hypoglossal nerve.

FIG. 6.—Granulated and swollen nerve fibers. (A b c), unequally enlarged axis cylinder; (d d d), transverse section of the same.

FIGS. 7, 8.—Amyloid degeneracy. (A), uncolored, (b), colored granules, with iodine.

FIG. 9.—Hypertrophied gray nerve fibers.

FIG. 10.—Neuroglia cells, with many processes.

All lesions of the medulla oblongata are not necessarily fatal. Some limited hæmorrhages may exist in the nuclei of the hypoglossus, facial, or a little higher up in the nucleus of the coculomotor nerve. Such affections will be marked by disturbances of the function of phonation, deglutition, certain forms of aphasia, or by strabismus. In injuries of the back of the head such symptoms are most frequent. (*Hallopeau.*) Lesions of the medulla oblongata produce disturbances in the visceral functions. Thus, may an injury in the floor of the fourth ventricle produce *polyuria*, *albuminuria*, *diabetes*, as *Claude Bernard* has demonstrated by his famous experiments of *Piquure* in that region. Modifications and disturbances of circulation, respiration, and temperature usually follow lesions or injuries of that organ. Injuries and lesions of the spinal marrow are very often followed by diseases of the medulla oblongata, and *vice versa*.

The Spinal Marrow. Anatomy.

The nerve bundles which pass through the nerve roots are connected with the ganglionic cells of the spinal marrow, partly through the prolongations of the axis cylinder (the *anterior cornua*), partly through the fine networks of nerve fibres (*posterior cornua*) to the formation of which they both contribute. From the gray substance nerve fibres enter into the adjoining white cords, which either unite portions of the gray substance situated below with those above, or ascend directly into the brain. The longitudinal fibres are arranged in bundles, or *fascicles*, according to their specific functions, and form the *columnar tracts*. Those constituting, in their course, the *anterior* and *lateral pyramidal tracts*, the *cerebellar lateral*, the *cords* of GOLL, and the *cuneate cords* are best known. The pyramidal lateral and anterior *columnar tracts* (*Turk*) contain peripherally-conducting fibres, which directly connect the gray spinal marrow substance with the cortex of the cerebrum (parietal and frontal portions). Passing through the cerebral peduncles and into the internal capsule, the lateral fibres cross in the pyramids to the opposite side, but the anterior descend on the same side into the anterior commissure, then cross and enter into the anterior cornu. The anterior tract is situated in the middle portion of the anterior column, the lateral in the posterior part of the lateral column. Their diameter decreases with their increased distance from the pyramids; they are not alike on both sides, and thus make the medulla spinalis an asymmetrical organ. The anterior cords are usually lost in the middle of the dorsal portion of the spinal marrow, but sometimes reach down to the lumbar region. The cerebellar tracts connect the gray spinal substance (especially the columns of CLARK) with the cerebellum. They usually reach the lowest dorsal portion. The other anterior cords are considered by *Flechsig* as mixed lateral tracts, uniting several portions of the gray substance with those at the base of the brain, and forming fibres of the roots of the nerves. The cords of GOLL (*funiculus gracilis*) connect the posterior roots of the spinal marrow with the tegmentum of the medulla oblongata, the Thalamus opticus and the corpora quadrigemina. The cuneate cords contain fibres which unite several parts of the gray substance with the tegmentum and the cerebellum.

The gray substance of the spinal marrow contains reflex centers, subordinate to those of the medulla oblongata. By irritating sensory fibres leading to its different portions, single and double muscles, and co-ordinate groups of these, in the body, may be put into motion. Sensations carried through the spinal centers into the brain there become perceptions. On the other hand, reflex actions from the spinal centers may be checked by the brain, and converted by it into voluntary muscular movements. (*Goll, Deiters, M. Schulze, Gerlach, Leyden, Huguenin, Boll, Schieferdecker, Eichhorst, Fleschig, Erb, Schwalbe, Bramwell, Kohler, Laura.*)

Lesions and Diseases of the Spinal Marrow. Hyperæmia, Hæmorrhage, Anæmia.

The quantity of blood in the spinal marrow and its membranes differs very much according to its state of rest or activity. Congestive hyperæmia only takes place either when the action of the heart is enormously increased, or when the resistance in the arteries and their branches in the membranes and the spinal marrow is abnormally diminished. Static hyperæmia is caused by obstructions in the venous current either in the spinal or in the cranial cavity, conditions existing in diseases of the heart or lungs. Local disturbances of arterial or venous circulation in the spinal marrow or its membranes give rise to local hyperæmia or congestion, as in tumors, exudations, etc., in the spinal canal. In the meningiæ hyperæmia is marked by great turgidity of the vessels, the smallest of which often become plainly visible when the quantity of blood is very great. In the white substance of the marrow, when the veins are extremely congested, the vessels are found exceedingly full, the gray matter assuming under such circumstances a dark, grayish-brown appearance, alternating with normal gray spots. Anæmia is marked, post mortem, in the meningiæ by thinness of the vessels and paleness of the membrane; in the marrow substance by opacity of the white and very pale gray color of the gray matter. Rachitic anæmia may be only a part of general bodily anæmia, or may be caused by vascular obstruction within the spinal canal or of the circulation connected with it. According to the extent of this stoppage of circulation the anæmia will be local or diffuse. Hæmorrhages are as frequent in the spinal marrow as in the brain. They may be caused by diapedesis or by rupture of the vessels. They may take place in the subdural or subarachnoid spaces. When they are light and confined to small spots there is hardly any destruction of the medullary tissue, but they leave behind pigmentary infiltrations. Not only acute and chronic diseases and lesions of the blood vessels and the heart, but also many infectious and miasmatic diseases are capable of producing thrombi and emboli in the meningial vessels or cause spontaneous ruptures of the same. Œdema of the spinal marrow consists of a state of thorough saturation of its tissues and its membranes with serous liquid. A cut surface of gray or white matter, in such a state, has a glassy, glistening appearance.

The perivascular sheaths, as well as the substance of the spinal membranes, may undergo dropsical alterations; the spinal canal may be filled with liquid (spinal dropsy). Accumulation of liquid within the cerebro-spinal cavities is chiefly due to arrest or hindrance of venous circulation. Acute œdema may suddenly take place when the heart's action is suddenly checked or weakened to a high degree. In enteric typhoid (*Buhl*), in thrombosis of the veins of the dura mater (*Krapelin*), in many heart and lung diseases, there may be formed venous stasis and acute œdema within the spinal canal.

Syringo-myelitis is a most singular alteration, taking place within the spinal marrow. It consists of excavations or formation of cavities, situated in the gray commissures behind the central canal and extending from there upward for quite a distance. Occasionally the cavities extend into the posterior cornua, which dilate sometimes posteriorly, sometimes laterally. The anterior cornua are very seldom thus affected. The affection may exist at any height of the spinal regions. The cavities are lined with neuroglia, and are filled either with a jelly-like transparent substance or with a clear liquid. The proliferation of the neuroglia, which precedes the formation of those cavities, begins within the central canal. Very rarely does it originate in the gray or white substance of the spinal marrow. In regard to the causes of these alterations the authorities differ. *Westphal, Schulze, Simon* attribute it to decay and destruction of the proliferating neuroglia preceding the excavations. *Langhans* finds that formations of sacs or dilatations of much distended blood or lymph vessels in the central canal are first formed; these lead to stagnations of the venous current and development of local dropsies in the central canal region. *Leyden* thinks it due to congenital hydrorachis and insufficient closure of the central canal.

Pathology and Symptoms of Spinal Hyperæmia.

Erb (*Diseases of the Spinal Marrow, V. Ziemssen's Hand. f. pathol., etc.*, p. 217) states: "Notwithstanding the great assurance with which many are inclined to speak of hyperæmia and congestions of the spinal marrow, their symptoms are still very indefinite and obscure; their diagnosis subject to many doubts and difficulties."

TABLE II.

FIG. 1.—Capillary emboli in the spinal marrow. (Ab), natural size; (c), magnified; (e), two small embolic spots, of a cellulo-granular kind.

FIG. 2.—Senile atrophic processes in the spinal marrow. (A), *pons varoli*, transverse section, several cicatrices and cysts; (b), section at different heights, atrophy of the posterior lateral cords in the upper enlargement; (a b), t. 1, 3, small sclerotic spots.

FIG. 3.—Hæmorrhage in left side of the fourth ventricle, in pons varoli and medulla oblongata. C. B., a man forty-nine years old, after a fall became affected with temporary paresis of the right arm and light aphasia. Two years later supervenes an apoplectic attack with alternate paralysis and complete anarthria, difficulty of swallowing, peculiar strabismus, hiccough, Cheyne-Stokes respiration, rise of temperature, and death. Autopsy: Cardiac hypertrophy and Bright's disease of the kidneys. Recent hæmorrhage in right corpus striatum. In the fourth ventricle an old copious extravasate penetrating into the medulla oblongata and passing thence into the cerebellar peduncle and lower portion of the pons. (*Leyden, Klinik d. Rueckenmarks-Krankheiten*, page 65, Part II.)

FIG. 4.—Cerebellar Tumor. It displaced and softened the medulla oblongata. (C), cerebellum; (P), pons varoli; (T), tumor.

FIG. 5.—Transverse section of spinal marrow softened by compression. The whole substance has a flocculent and granular surface. Its gray portion is plainly visible.

Fig. 6.—(A, b, c, d, e, f), several transverse sections of the spinal marrow in several regions of its extent, showing the different alterations taking place in disseminated sclerosis.

FIG. 7.—Traumatic myelitis in the lumbar region. (A), grayish-red softening; (b, 1, 2, 3, 4), transverse sections (after the marrow substance has been hardened and stained with carmine and turpentine): (1) secondary degeneration of the cords of GOLL, (2) softening, (3) same as in (a), (4) portion of filum terminale, in atrophy of the gray anterior cornua; (c), sclerotic portion of the atrophied anterior cornua.

FIG. 8.—Elements, in a sclerotic portion of the spinal marrow, in softening myelitis. (A), blood vessels covered with granular cells; (b), nucleus of neuroglia; (b′), fat molecules; (c), fibers of neuroglia in a state of fission; (d), coarse fibers of neuroglia in cell-production.

FIG. 9.—Central Myelitis. (A b c), section showing the lesion.

FIG. 10.—Nervous Elements in Myelitis. (A b), swollen spheroid ganglionic cells; (cc), such as contain vacoclæ; (d d), transverse section of nerve fibers in a state of cell division and reproduction.

FIG. 11.—A small cyst in the gray matter of the right anterior cornua. Atrophy of its ganglionic elements.

The most prominent symptoms are sensory irritations. There is a dull pain over the whole back, with a sensation of pressure upon the organ. It is not very violent, and does not increase by pressing upon the spinous processes of the vertebræ. Formication, itching, and stinging, etc., with violent pain, are felt in the extremities—especially the lower—soon after the attack. The skin becomes more sensitive, and reflex irritation is increased. Girdling sensation (*Hammond*), transitory light muscular spasms, twitching and tremor also exist occasionally. Electric irritability of the muscle is much increased (*Rosenthal*). A little later symptoms of depression make their appearance. The lower extremities feel heavy, often perceptibly insensible. Muscular weakness, light paresis, and fatigue after the slightest effort are then manifest. In simple congestion total paralysis never exists. The above symptoms are generally bilateral. They are either confined to the lower extremities, or have there their beginning. When extending to the upper extremities it takes place very speedily. The pain, fatigue, etc., pass from one part of the body to another, seldom remaining stationary in one locality. *Brown-Sequard* claims that the symptoms increase in severity in the recumbent position, but decrease in the erect posture; that rest aggravates, and motion mitigates them. There is never any fever in uncomplicated spinal hyperæmia. The pulse may be slow or frequent. There is more or less disturbance of the general functions of the body. When no untoward complications supervene the disease terminates in recovery.

Clinical Symptoms of Spinal Hæmorrhage.

According to the extent and intensity of the extravasation of blood the disease-phenomena will differ. In severe cases the person is suddenly seized with very violent pain, and without losing consciousness breaks down at once. Or he may go to bed at night, apparently in good health, and wake up completely paralyzed. At other times there are prodromal symptoms of spinal congestion for days and weeks, or of acute central myelitis (such as feeling of uneasiness, fever, violent pain, formications, girdling pains, heaviness of the limbs or numbness, trouble of the bladder), which may last several hours or even days, and then the sudden paraplegia will appear, usually accompanied with either local pain or extended over the whole spine, but which disappears shortly after the stroke. The paralysis may affect only the upper portion of the spine, the lower, or both. Dyspnœa and other respiratory troubles attend paralysis of the upper part of the spine, because the respiratory muscles are unable to perform their functions, and complication with troubles of the circulation will soon supervene. With the muscular paralysis there is usually connected anæsthesia, and nearly always paralysis of the rectum and bladder. Reflex phenomena vary according to the seat of the lesion. When the gray substance is totally affected there will be total absence of reflex action, but when only parts are injured reflex phenomena may still continue in some localities. The lesser the extent and depth of the hæmorrhages, the slighter are the morbid disturbances of function, and, also, the more insignificant the results. Some light forms of spinal hæmorrhage pass entirely unnoticed. The only traces left of these are pigmentary spots found in the marrow after death. Consciousness, even in the severest forms, if uncomplicated with brain trouble, is never lost. Pain in the back sometimes exists, but there is no tenderness on pressure upon the spinous processes. Muscular spasms are rare, and only affect the unimpaired muscles. Formication, tingling sensations, and numbness are very often experienced. The results of such sudden severe attacks are soon manifested by speedy formation of gangrenous bed-sores on the trochanters, the sacrum, the heels, and on all parts of the body subject to pressure. These sores are not only a source of trouble, but also of danger to the life of the patient. Secretion of the urine is soon altered; it becomes bloody, purulent, albuminous; the alkalinity of the urine in the paralyzed bladder develops catarrhal cystitis and pyelitis. Fevers follow, chills, rigors, hectic, pyæmic or septicæmic exacerbations; gangrenous decubitus and suppurative cystitis, often rectal catarrh soon destroy the life of the patient. Muscular atrophy and other degenerations of the tissues are very rapidly developed. Contraction and deformities in the joints are produced in slower progress of this disease. Reflex phenomena are gradually lost. *Hayem* names a chronic form of spinal hæmorrhage, in which it took place in an already affected spinal marrow, and quotes cases described by *Nonat*, *Massot*, and *Lancereux*, answering to his definition of the disease.

Acute Myelitis. Lesions Located in the Gray Substance. Ganglionic Affections.

Infantile paralysis, as first described by *Duchenne* in 1864, is usually an affection of children from the ages of one to five years. The attack is nearly always sudden, beginning with a very violent fever, coma, delirium, convulsions, and very speedily develops complete muscular paralysis and absolute weakness in those structures. It has generally the form of paraplegia. There is hardly any noticeable disturbance of sensation or paralysis of the rectal or vesicular sphincters. Speedy recovery of the general health soon follows, with gradual improvement in the lamed parts, yet unequal recovery of motion; parts of the limbs remain permanently paralyzed. In some of the muscles there is formed a rapidly progressive atrophy and degeneration. The growth of the bones generally remains stationary, the flesh of the limbs is cold and looks blue. Deformities of the trunk and joints, contractions and distortions of some individual limb or parts of limbs gradually form. The general health becomes good. The preponderating number of this class of paralytics being among children has given it the name of infantile paralysis; but the same may befall persons of any age, and the acute symptoms of the attack and the resulting clinical phenomena may vary exceedingly. The anatomical lesion is inflammation and subsequent degeneracy of the anterior gray columns. The affection may happen at any level of the spinal marrow, but the gravest forms are usually found in the cervical and lumbar enlargements. The several authors have assigned different causes to the disease. The older clinicists (*Lange*, *Heine*, etc., even *Duchenne*, the elder) have ascribed it to the irritation existing in the process of dentition. *Wharton Sinkler*, who reports having examined 57 cases, found not less than 47 of them to have been brought about by atmospheric influences. In very numerous cases some connection seemed to have existed with previous infectious diseases from which the patients had lately recovered.

The researches of *Vulpian* and *Prevot*, in 1866, *Joffroy*, *Charcot*, *Damaschino*, and *Rogers*, have demonstrated that there is atrophy of the motor cells of the anterior cornua of the spinal marrow in this disease. That when the muscular paralysis happens to exist only in a confined group, it is because only a limited number of motor cells are atrophied and degenerated. (*Michaud* and *Pierret*.) Not only are the multipolar ganglionic cells completely degenerated, but also the axis cylinders and nerve fibres have undergone a similar change, and are never restituted. This explains the permanent character of the motor paralysis and the subsequent muscular atrophy. Although general sensibility (with the exception of the first stage of the attack) remains absolutely undisturbed, yet reflex irritability is completely destroyed in the affected portions, and to some extent in the unaffected parts, especially in the early part of the disease. A milder form of this anterior polio-myelitis is described by many authors (*Duchenne*, *Volkmann*, *Frey*), and named by *Kennedy* temporary paralysis. Whether the two are the same or not the future will decide.

Acute Central Myelitis.

This form is an acute inflammation of the spinal marrow, in which the lesion is developed in the gray central axis, and appears in disseminated and irregular spots. The central ependymic canal is the seat of a very severe catarrhal inflammation of its epithelium; the adjacent gray substance, the central, the anterior and posterior cornua, the commissural gray matter thereby become very much affected. This lesion has a more general than a localized inflammatory character. The many hæmorrhagic points found in the marrow substances are, according to *Hayem* (These agg. 1872), a form of red softening, which *Leyden* (*Klinik d. Rueckenmarks*) compares to red hepatization of the lung in primary stages of pneumonia. Its symptoms are, as may be expected from a general inflammatory condition of the spinal marrow, usually very severe and manifold. High fever, disturbance of sensation (formication, tingling, prickling, etc.), and soon gradual muscular paresis is followed by complete paraplegia, extinction of reflex phenomena. The rectum and bladder become paralyzed, muscular atrophies, gangrenous decubitus, etc., follow in rapid succession. When the inflammation is confined to a certain locality, the white or gray substance, to a certain level only, the clinical features will vary according to the locality, the intensity of the morbid processes, the concomitant alterations in the acute stage, and those following in the further development of the lesion. Muscular paralysis, with atrophy, disturbances of sensation and sensibility, visceral derangements, trophic disturbances, etc., will result from injuries, alterations, destructions, or obliterations of the specific spinal centra of innervation, in the organs of sensation, motion, respiration, circulation, secretion, and excretion of the body.

Transverse myelitis (that is, when the acute inflammatory process extends transversely upon the gray and white substance, which usually happens in traumatic lesions of the spinal marrow) is the least promising and the most dangerous of all forms of myelitis, for the consequences of such widespread functional disturbances speedily lead to death.

CAPILLARY EMBOLI.

Fig. 1.

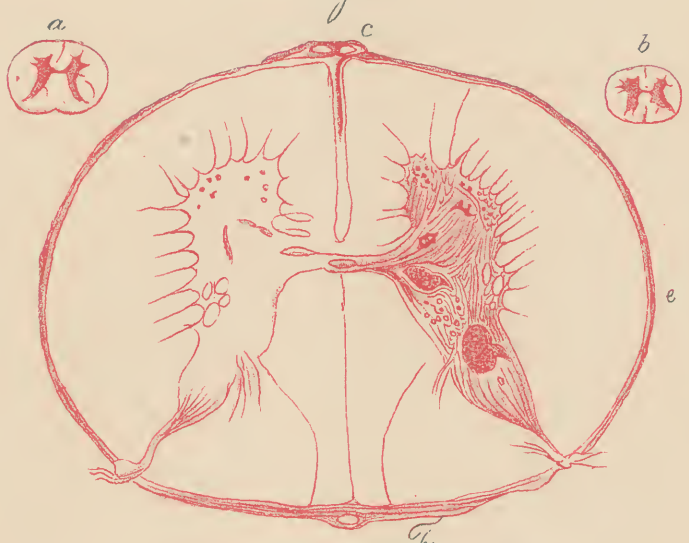
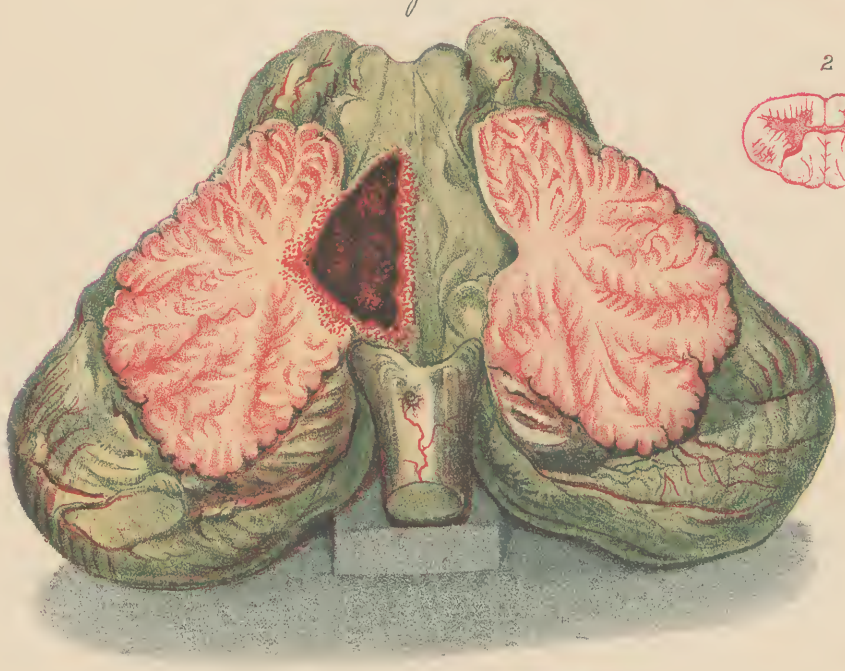
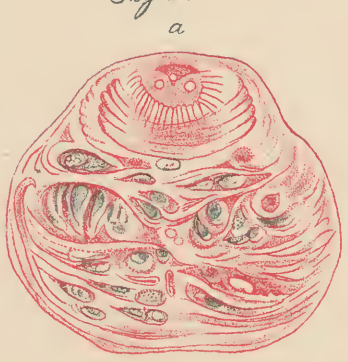


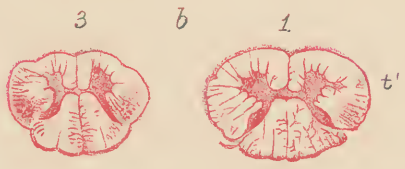
Fig. 3.

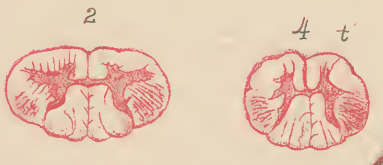


CEREBELLAR TUMOR.

Fig. 2.







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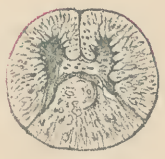


Fig. 4.

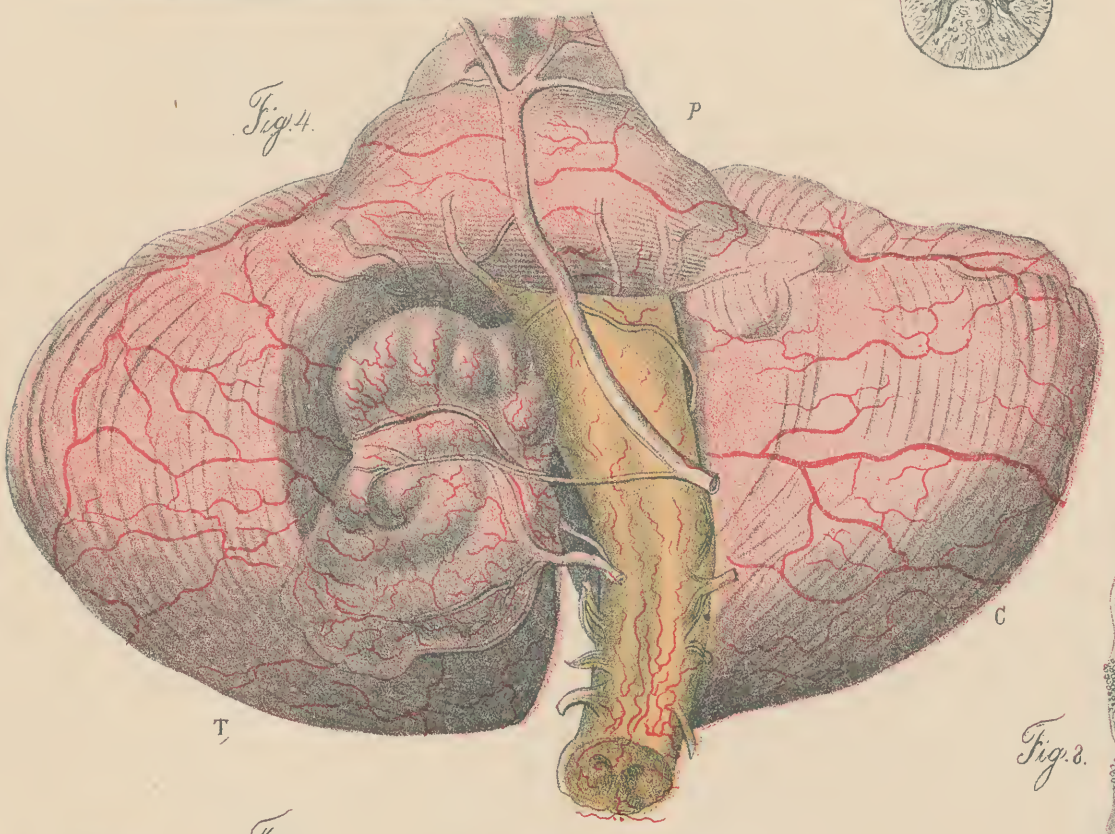


Fig. 9.

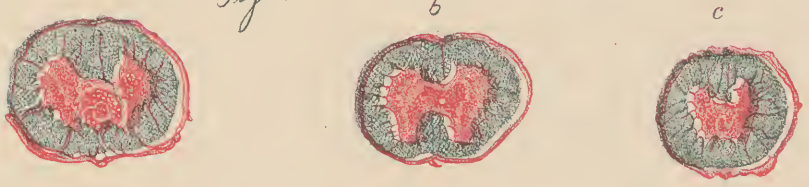
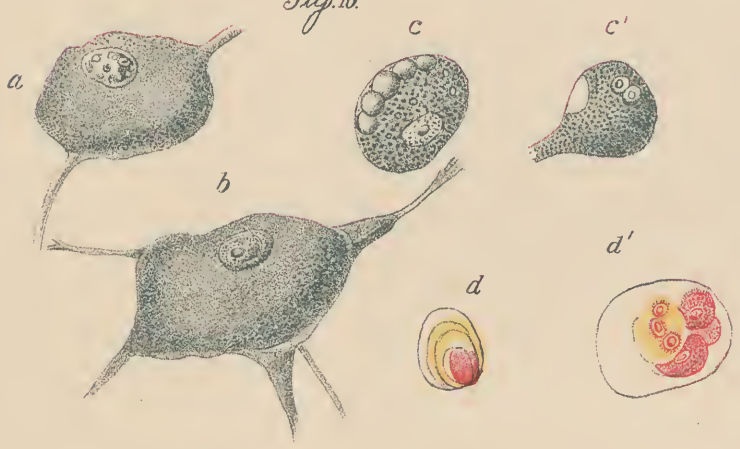


Fig. 10.



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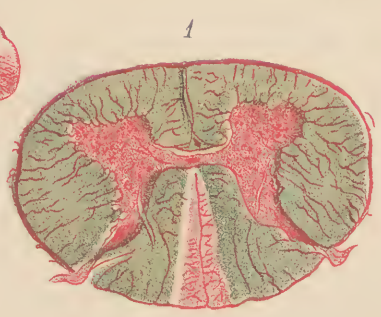


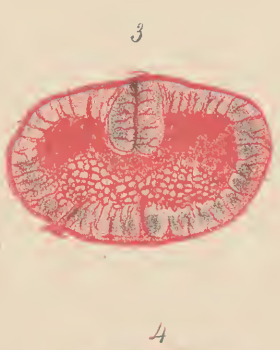


Fig. 7.

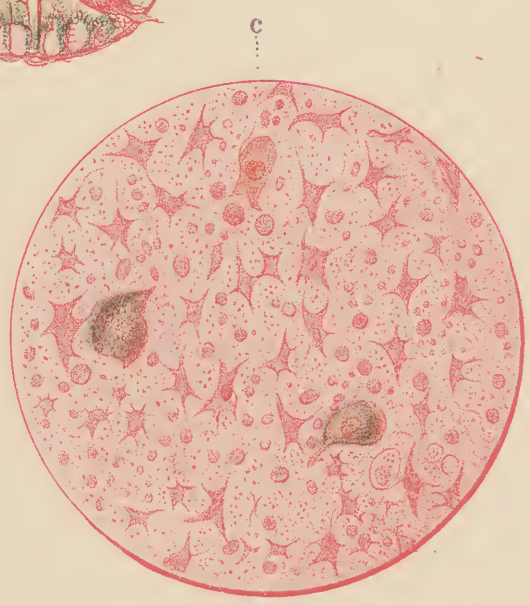


Fig. 8.

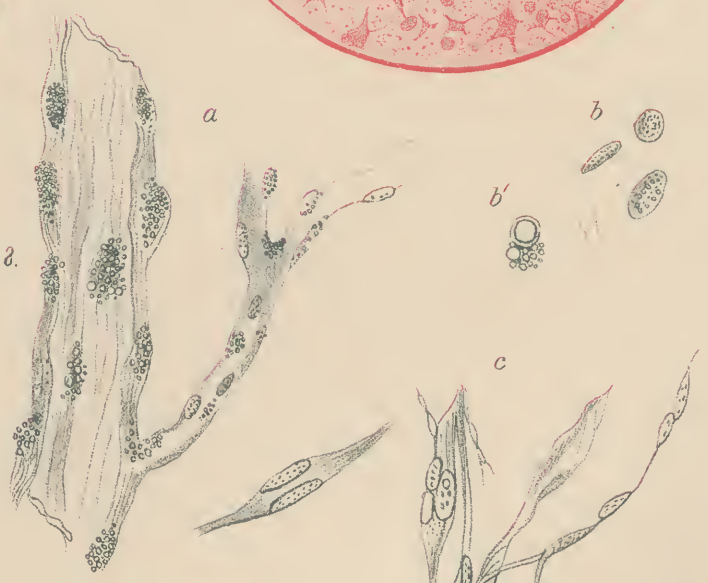
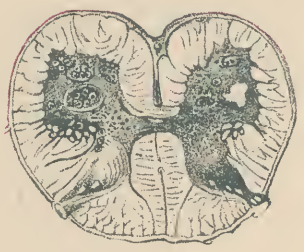
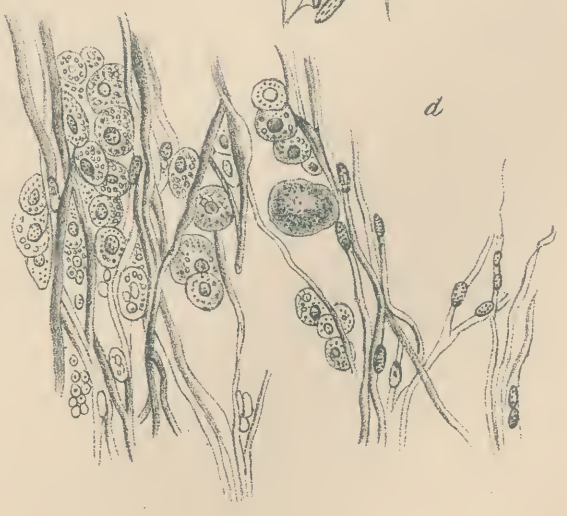


Fig. 11.





SCLEROSIS OF THE POSTERIOR COLUMNS OF THE SPINAL MARROW.

Sec. V. *Tab. III.*

Fig. 1.

Fig. 2.

Fig. 2. a. b.

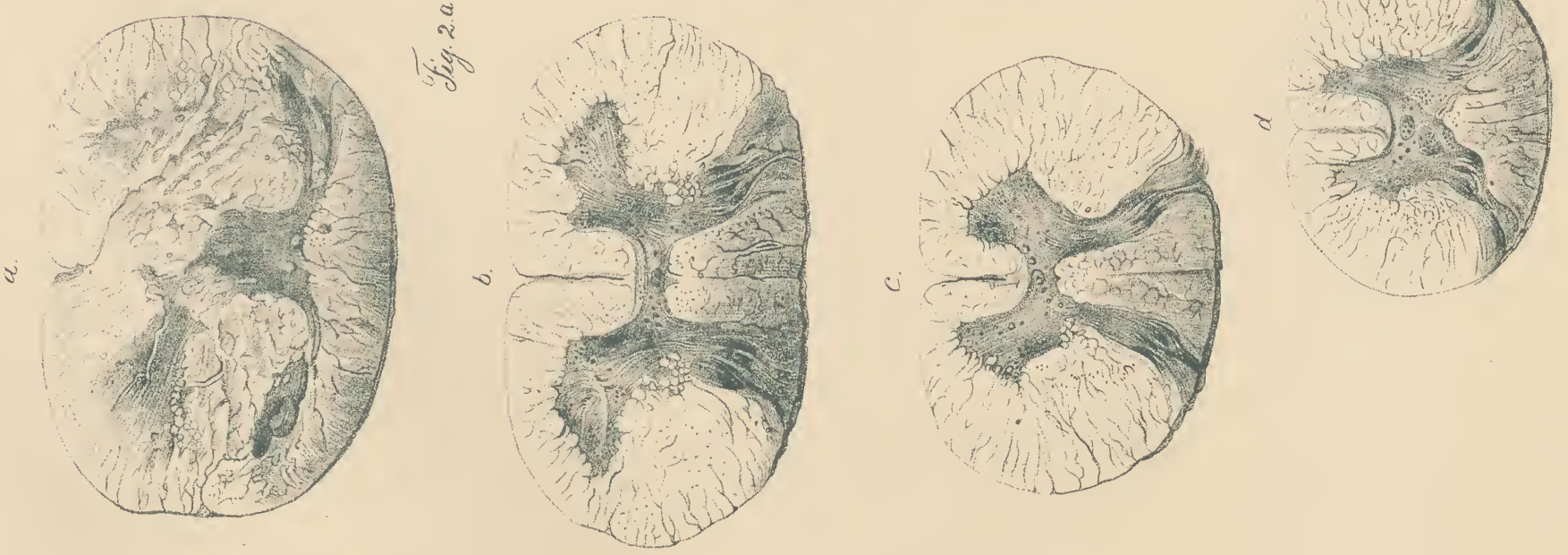


Fig. 2. a.

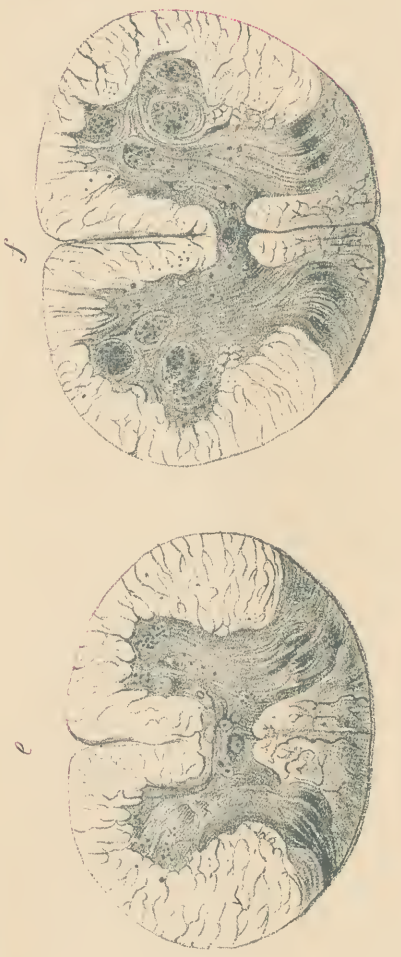


TABLE III.

FIG. 1.—*Sclerosis of the Posterior Columns of the Spinal Marrow. Tabes Dorsalis. Progressive Locomotor Ataxia of Duchenne.*

The spinal marrow is here represented in its natural size; the posterior aspect showing the gray degeneration of the posterior cords, as seen in the transverse sections (fig. 3) a, b, c, d, e, f, made in several parts of the organ. The posterior cords are shortened, and the degenerative process increasing in extent from the periphery toward the middle. There is distinct atrophy of the radical fibres and the columns of CLARK. (The cuts are magnified five diameters.) The case, as reported by *E. Leyden*, is as follows: The affected person, an officer in the German civil service, was a man thirty-one years of age. He was a robust, muscular man, with a very healthy look about him. Had manifested for some years a number of important ataxic symptoms, such as numbness in the lower limbs, inability to co-ordinate some certain movements, etc.; but having undergone some treatment and obtained a little benefit therefrom, his general health also being quite good, he undertook a very hard journey to Russia in the heart of winter. Severe fatigue and hard official work, also the great severity of a Russian winter, compelled him to return home, but in a very broken-down condition. His gait became very laborious and shuffling; with closed eyes he is unable to perform any movement requiring co-ordination of muscular groups. In the lower extremities there is partial anæsthesia; however, pricking with a needle is readily perceived. In the upper extremities the symptoms are very light. A variety of treatment was undertaken by his attending physician with but little benefit. A year before his death he contracted a very severe pneumonia, from which he recovered almost altogether, but the spinal trouble continued uninterruptedly. About six weeks before death both kneejoints became much swollen, and finally an abscess developed in the left thigh and caused burrowing of pus in the depth of the muscles. The abscess was opened by a wide incision and anti-septic drainage carried out. A number of metastatic abscesses formed, nevertheless, in both lower extremities and eventually produced septic cachexia, which soon destroyed his enfeebled health, and he died of a consumptive fever.

Autopsy (made by *Prof. E. Neuman*) the day after his death: The spinal column and spinal dura mater showed nothing abnormal. The pia mater thickened and opaque posteriorly, the gray alteration of the whole length of spinal marrow readily seen through it. The organ is flattened in the cervical region; the gray degeneracy visible in the cervical, dorsal, and lumbar regions. In these regions the radical cords are thinner than the anterior. The lateral columns normal. Many nerves of the cauda equinæ have a gray color. In both knees marks of gangrene of the abscessed tissue.

FIGS. 2, 2*a*.—*Chronic Myelitis.* Sclerotic alterations of the spinal marrow, disseminated in several regions of its length. Figs. 2*a*, 1, 2, 3, 4, 5, 6, 7 are a number of transverse sections of the organ showing the extent of the lesion in different localities. The tissue was hardened and stained with turpentine and carmine before examination. At 2*a-b* several small cysts are seen in the white matter; the other sections show the extent of the alterations in the gray substance.

Chronic Myelitis. This comprises all slowly-developing, insidiously-invading, feverless, and long-protracted morbid processes in the spinal marrow; also those slowly developing alterations in its different tissues, consequent upon acute myelitis. They may consist of indurative inflammations, sclerosis, gray degenerations, and even slowly-produced softening. The lesions may be confined to definite localities or diffused over the whole organ, assume a transverse or a longitudinal direction, occupy quite a considerable portion of the spinal marrow, or be confined to small portions, or affect the gray and the white substances at the same time or one kind only. To the naked eye the diseased part appears gray, translucent, shrunk below the level of the healthy tissue, yet not clearly demarked. It is of greater consistence than the normal tissue, and sometimes tough and dry, often soft and succulent. In the great variety of processes and forms of lesion comprised within the scope of chronic myelitis it is to be expected that a corresponding variety of clinical symptoms should exist. The most frequent form, *i. e.*, chronic transverse myelitis, may serve as typically representing the chief clinical features.

The symptoms develop slowly, one after another, and corresponding to the different centra situated in the different levels of the marrow. These are: girdling sensation, pains in the back, motor and sensory paralysis; then follow sphinctral paresis or paralysis. Reflex irritation is usually increased. More or less severe paraplegia. The general health is usually good, but in the later, and especially in the last stages of the disease, it becomes aggravated by catarrh of the bladder and annoying and painful bed sores. The muscular atrophies following in the several paralyzed limbs are due to the lesions and degeneration of the motor cells and other portions of the gray substance in the anterior cornua. The degenerations are either primarily or consecutively developed.

To the first type, *the progressive muscular atrophy*, as described by DUCHENNE and ARAN, and clearly set forth by CHARCOT in his *Leçons Cliniques*, must be counted. The most prominent feature of this affection is the gradually (and without any premonitory symptoms) developing atrophy of single or groups of muscles in one or the other limb, or in one or the other part of the body. Sometimes one or more muscles of the hand, the forearm, the arm or shoulder gradually begin to waste away, one after another in succession. Or the same atrophic process may go on in a lower limb and then stop; or in the trunk and extend no farther. Close examination shows that the destruction progresses from fibre to fibre. With the muscular waste there is connected a greater or lesser disability of motion. Yet the paralysis is never so complete as in paralytic troubles originating in the brain. Deformities consequent upon the changed positions of the limbs or part of the body, as the case may be, will gradually form. When the diaphragm or other respiratory muscles are thus attacked in their entirety, disturbed respiration and very dangerous complications will ensue. The muscular tissue undergoes fatty and granular changes from lack of innervation. When chronic inflammations spread from the adjoining portions of the spinal marrow into the anterior motor nerve-cells, secondary muscular atrophy sets in. The spread may be transversely from the meningiæ or the white columns into the anterior gray cornua. The muscles become very often atrophic before the total alteration in the centra.

Tabes Dorsalis. Sclerosis of the Posterior White Columns. Locomotor Ataxia.

These are synonyms of a spinal lesion, occupying a definite transverse position, but having an indefinite extent in the longitudinal direction of the organ. It is *Friedreich's gray degeneration* or *degenerative atrophy of the posterior cords.* This lesion was partly known to and described by Hippocrates and Galen as a form of spinal consumption. Only since 1827–32 has its anatomy been sufficiently studied by *Hutin* and *Monod*, and a number of fine illustrations with clinical histories of several cases were given by *Cruveilhier* in his celebrated Atlas of Pathol. Anatomy. Since then the writings of *Horn*, the classic description of *Romberg*, who detailed the most prominent symptoms of the disease, the anatomical researches of *Froriep*, *Jacoby*, and the pathological studies of *Steinthal*, *Bouillaud* and *Todd*, *Rokitansky* and *Turk*, have well-nigh defined the pathological character of tabes dorsalis. In 1858 *Duchenne*, after exceedingly careful observations of its clinical phenomena, characterized it as a disease in which disturbance of co-ordination (ataxia) forms the central point, but erroneously located the lesion in the cerebellum. He named it *ataxie locomotrice progressive.* Since then every phase of the disease has been investigated and thoroughly elucidated. Great numbers of treatises on the subject of tabes dorsalis have been and still are accumulating. (*Trousseau*, *Dujardin*, *Mar. Carre*, *Bourdon*, *Luys*, *Oulmont*, *Teissier*, *Dumenil*, *Charcot*, *Vulpian*, *Jaccoud*, *Friedreich*, *Eisenman*, *Leyden*, *Westphal*, *Spaeth*, *Remak*, *Lockhart Clark*, etc.

Pathologically considered it is a form of chronic myelitis, mostly affecting the young and middle-aged. Its morbid alteration is characterized by band-like, gray, sclerotic degeneration of the posterior white columns, consecutively involving the adjacent parts of the lateral white and posterior gray columns. The affection usually begins in the lumbar region and extends upward even into the medulla oblongata.

Clinical Phenomena. The first stage, which may last many years, is characterized by lancinating pains, cerebral disturbances (trouble of the optic and other nerves of the eye), paræsthesia in the lower limbs, the trunk, in the forearm, weakness and lassitude and unsteadiness of gait, troubles of the genito-urinary organs, etc.

The second stage or pathological phase, gradually passing from the first, is marked by the appearance of certain cerebral nervous derangements, and chiefly by characteristic disturbance of co-ordination of movement or locomotion, without, at first, any kind of muscular weakness, degeneration, or loss of electric irritability. The sensory disturbances of the first stage usually remain, to a greater or lesser extent. As a rule, there is a third stage to this disease, which, coming on slowly, is characterized by progressive muscular paralysis, atrophy, and contractions, catarrh of the bladder, bed sores, and general marasmus. There are sometimes long intervals between the several phases, with corresponding cessation of some morbid processes. Here and there the pause is very long, especially in the early period of the disease. The patient may then improve for quite a while. Recovery from this disease is very rare.

Anatomical Character of this Lesion. A transverse section of the spinal marrow in the affected portion will very plainly show the degenerated posterior columns as a grayish-red gelatinous mass situated between the posterior gray cornua. The boundaries between these and the posterior commissures are obliterated. The mass is soft but elastic, not at all friable, and contains a few spots of unchanged tissue. The alterations may differ in different cases. Usually the whole length of the spinal marrow is thus affected. The posterior gray cornua and the radical fibers are always affected. At the base of the posterior cornua the columns of CLARK have a singular gray appearance. In some cases the degenerative process passes from the posterior into a large part of the lateral tracts, and in extreme cases even along the outer edges of the anterior white columns. The alteration may reach down to the filum terminale, or ascend into the brain, into the pons varoli. The posterior spinal nerve roots are never found involved.

Functional Disturbances. Although each of the muscles remains for years unchanged and under complete control of the will, yet when a group of them are to act in a co-ordinate manner their harmony is disturbed, and their conjoint action eventually becomes impossible. The muscles obey the voluntary impulse individually, but refuse conjugate action, so that a limb or a part of a limb of the body may be thrown from the track on the line of its movements, and all sorts of deviations from the equilibrium of the body, or the limb, may take place. The lower extremities are oftenest and most intensely thus affected; the upper limbs not so often nor so much. In uncomplicated cases the eyes and face are never involved.

Disturbance of Sensation in this Lesion. Pains of the most excruciating kind and deep-seated in the muscles, the joints, the bones, etc., come on suddenly, and often as suddenly disappear. They may pass like a flash, or continue for some time. The surface of the affected part is extremely sensitive to the touch. The trunk experiences a sensation as if tightly bound by a rope or belt (girdling sensation). Hyperæsthesia may alternate with anæsthesia in some localities, although anæsthesia oftenest exists. There is gradual loss of sensibility. Some impressions upon sensory surfaces may become painful, while others may pass unnoticed by the senses. In the affected muscles muscular sense is obliterated. The sense of

TABLE IV.

FIG. 1.—a, b, c, d, e, f, g. *Typical Case of Disseminated Sclerosis of the Spinal Marrow and the Mesencephalon.* Fig. 1 is of natural size, and shows a number of gray spots in several regions of the spinal marrow, the medulla oblongata, the pons VAROLI, the left crus cerebri. Fig. 1, a, b, c, d, e, f, g, shows a number of sections, hardened and stained. The predominant lesion and shrinking in the cervical region is readily noticeable.

FIG. 2, a, b, c. Progressive bulbar paralysis and muscular atrophy. A, section of the medulla oblongata; b, cervical region; c, cervical enlargement (much magnified).

FIG. 3, a, b, c, d, e, f. Transverse sections of the spinal marrow (magnified slightly) in different regions of the organ in a case of typical disseminated sclerosis. In the region of the cervical enlargement (a) the alteration is least; in the region of the lumbar enlargement (e) it is most prominent.

FIGS. 4a, 4b. Transverse section of the spinal marrow in a case of infantile spinal paralysis. In both figures the alterations exist in the motor ganglionic cells of the anterior gray cornua. A, b, alteration in the cervical regions. *Seligmuller* and *Leyden* (in *Klinik d. Rueckenmarks Krankheiten*) describe each a case of bilateral infantile paralysis. The latter found a child of 10 years of age whose whole upper extremities were paralyzed from the time of its birth. The child had undergone artificial delivery, and the attending accoucheur stated that he used traction of the feet of the child whilst its arms and head were engaged in lower strait, for quite a while, when he thinks straining of the spinal marrow in the cervical region took place. Both upper extremities were in a perfect state of atrophy.

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sight has to compensate for the loss of the muscular sense in the movements of the patient. With closed eyes he is utterly helpless, and unable to perform any kind of co-ordinate movement.

The lesion constitutes a progressive loss of co-ordination. Its exact locality is not yet determined. A number of complications with other lesions located in the adjoining structures of the spinal marrow, which invariably take place in the progressive stages of the disease, render the exact definition of this lesion very difficult.

Amyotrophic Lateral Sclerosis.

This is a chronic form of myelitis located in the lateral white cords of the spinal marrow. *Charcot* and *Gombault* were the first who described and named it. In the typical cases reported by the first author, the lesion was confined to the lateral white cords, without any complication in the gray matter, which sometimes happens in this disease. A transverse section of such an affected part shows the sclerotic alteration to extend to the external angle of the anterior cornu, while posteriorly it reaches the posterior cornu. Outside it is separated from the cortical layer of the spinal marrow by a band or zone of unaffected tissue. The symptoms chiefly characterizing this lesion are: Paresis of the limbs, followed by deformities and contractions. When the upper limbs become affected the hands soon become deformed. When the lower limbs become involved enormous muscular contractions and deformities follow the paretic state. When lateral sclerosis extends into the anterior cornua secondary atrophy follows the muscular contractions. According to *Charcot* the disease usually begins in the upper limbs, which speedily lose their power to move. Then muscular atrophy follows; the arm becomes stiff, and is tightly fixed to the trunk; abduction from the body causes severe pain in the shoulder. Or the forearms become flexed at the elbow, and every attempt at extension is extremely painful. The same may be the case at the wrist, etc. The neck may become stiff and immovable in any direction. The lower limbs may become similarly affected, deformed, and unable to support the trunk; erect posture becomes impossible, and finally general paralysis supervenes. All the paralyzed muscles waste away and become contracted. Any attempt at flexion of the extended limb or at extension of a flexed one produces rigidity of the whole member, followed by trembling. In the last stage, when the sclerotic process has reached the brain, the person succumbs to progressive bulbar paralysis. One of the chief characteristics of this lesion is its very rapid progress and speedy, fatal end.

Descending Degeneracy. Secondary Sclerosis.

This lesion is also located in the lateral cords of the spinal marrow; it follows upon lesions of the hemispheres, the crura cerebri, and the pons varoli. In the spinal marrow it constitutes a sclerotic alteration in the side opposite to that of the cerebral lesion (*Vulpian, Charcot, Bouchard*). It has neither the same extent in the lateral cords, nor does it occupy the same points, as in amyotrophic lateral sclerosis. According to *Charcot*, this degeneracy in the medulla oblongata comprises all pyramidal fibers. In the cervical region of the spinal marrow it occupies a small triangular space between the anterior and posterior gray cornua; in the dorsal region it occupies a smaller space and dwindles down to an insignificant spot in the lumbar region. In primitive lateral sclerosis it is diffuse and of great extent, implicating the anterior roots in front and reaching into the posterior cornua. In secondary sclerosis only a portion of the nerve fibers which make up the lateral cords are involved.

Clinical symptoms: (1) persistent paralysis of voluntary motion; (2) slowly developing into complete muscular atrophy—in about five or six months after the cerebral attack.

Diffuse Myelitic Affections.

Chronic transverse myelitis has been described. Myelitis from compression, as is produced in *Potts'* disease, vertebral or intra-spinal cancer or other tumors, is manifested by distributive disposition of symptoms, differing somewhat from those of the above. *Potts'* disease is characterized by spinal deformity, pseudo-neuralgic pains, paralysis or paresis of motion, or anæsthesia when the anterior or posterior cords have become more or less affected by the pressure exerted upon the dislocated parts. In vertebral cancer paraplegia and excruciating, painful hyperæsthesiæ are developed from compression of the spinal nerves by the altered vertebræ. Small rachitic tumors can not be diagnosed. Larger ones manifest different symptoms of compression, etc.

Disseminated Sclerosis in Plaques. (CHARCOT.)

This author recognized in this affection three principal forms: A cerebral, a cerebro-spinal, and a purely spinal. The most distinct characteristic of the spinal form is a peculiar *tremor*, differing from that of paralysis agitans and general paralytic tremor. It only takes place when the affected person performs voluntary movements having a certain intention or aim, such as walking, or carrying food or drink to the mouth. In perfect rest it ceases. It differs from the jerking and queer movements of chorea in this, that in disseminated sclerosis the tremor increases in intensity the nearer the purpose of the movement is fulfilled. In chorea the general direction of the movement is disturbed from the beginning by absolutely contrary motions, to an extent of frustrating the aim of the intentional motion. In chorea the tremor consists of gesticulations, jerks; in multiple sclerosis there are rhythmical shocks, oscillations. *Charcot* has observed in some cases lancinating pains, formication, and jerking in the lower limbs in walking, titubation when the eyes are closed. The following symptoms also characterize this multiple sclerosis: tremor of the extremities, impediment in speech, nystagmus. It is evident that very many symptoms will be found in this protean lesion characterizing many localized spinal affections. The cerebral form of this lesion is brought about by pachymeningitis, which is followed by thickening of the dura and pia mater, extending into the cervical portion of the spinal marrow. The sclerotic ring which surrounds externally the medullary cord compresses the roots of the nerves and produces more or less complete deformity of the organ. The functional disturbances follow in two stages: The first is manifested by pain and stiffness of the neck, formications and numbness, paresis and spasms of the muscles of the neck and upper extremities. The second stage follows with paralysis and subsequent atrophy of the upper extremities, with anæsthetic spots upon the skin. This is followed by contraction and a peculiar attitude of the hand, which *Charcot* has characterized as THE PREACHER'S HAND.

The symptoms in the three stages exactly represent the gradual evolutions of the morbid processes in the spinal marrow. (1) pain from compression of the posterior or sensory roots and cords; (2) paralysis and contraction from lesion of the lateral cords; (3) atrophy when the affection extends into the anterior gray cornua.

Diseases of the Spinal Meningiæ.

There is neither a disease nor a lesion of the spinal marrow but that one or all its membranes may not be affected. Only in the earliest stages of diseases of the spinal vertebræ are the membranes not involved; in all more advanced stages the dura mater either shares in the chronic inflammation going on in the osseous tissue or becomes infected with pus. When this happens the membrane will soon undergo morbid changes. Mechanical injuries of the spine, if at all serious, cause the membranes to partake in the lesions, which may consist of congestions, hyperæmia, hæmorrhages, or may be subsequent inflammations. Under such circumstances the membranes are really the most seriously involved. In acute and chronic diseases of the spinal marrow the membranes will undergo primary or secondary alterations. There are certain clinical phenomena, sometimes manifested in diseases of the spinal marrow, which give a peculiar character to the affections of the membranes, and may be considered, at least from a clinical point of view, as separate diseases. Such are the epidemic and some other spinal meningites. Generally speaking, the anatomical alterations, found in diseased spinal meningiæ, are singularly analogous to those of the cerebral membranes. Especially is this the case in acute and chronic inflammation of the pia maters and arachnoids of the two nerve centers. But there are essential differences nevertheless. Hæmorrhages in the cerebral arachnoid are usually derived from the dura mater or the interior of the brain; in the spinal arachnoid from the pia mater. The morbid processes peculiar to the sinuses in the cerebral dura mater do not at all exist in the spinal, for here no such class of vessels exist; but on the other hand there are many diseased conditions existing in the interlamellar space of the spinal dura mater, caused by disturbance of circulation of the vessels in that cavity, such as hyperæmia, congestions, or even hæmorrhages. In the connective tissue, so filled with fat surrounding the spinal cord, many forms of inflammations, suppurations, and exudates may find place. Hæmorrhagic pachymeningites are far more frequent in the spinal than in the cerebral dura mater. There are a number of morbid changes found on the spinal membranes which are not near as morbidly important as they were formerly thought to be. These are:

1. Ossifications of the dural membrane. Extensive ossifications are of the very rarest occurrence, while small ossoid plates are very frequent, and are utterly devoid of any causal connections with diseases of the spinal marrow. In chronic spinal pachymeningitis calcareous nodules are often disseminated over the dura.

2. Pigmentations of the membrane, especially in the cervical region, are due (according to *Virchow*) to deposits of coloring matter in the tissues of the membrane and not to past hæmorrhages or miliary infarcts, as it was thought formerly.

3. Hydrorachis. Analogous to external cerebral hydrocephalus is this more a congenital malformation than a consequence of active hyperæmia or congestion. The morbid symptoms present in that disease are due to incomplete development and lack of total closure of the walls of the spinal canal. There are, however, certain clinical phenomena of dropsical effusions in the spinal canal which are due to venous stasis, and have an actual morbid significance.

TYPICAL CASE OF DISSEMINATED SCLEROSIS, SPINAL AND CEREBRAL.

Fig. 1.

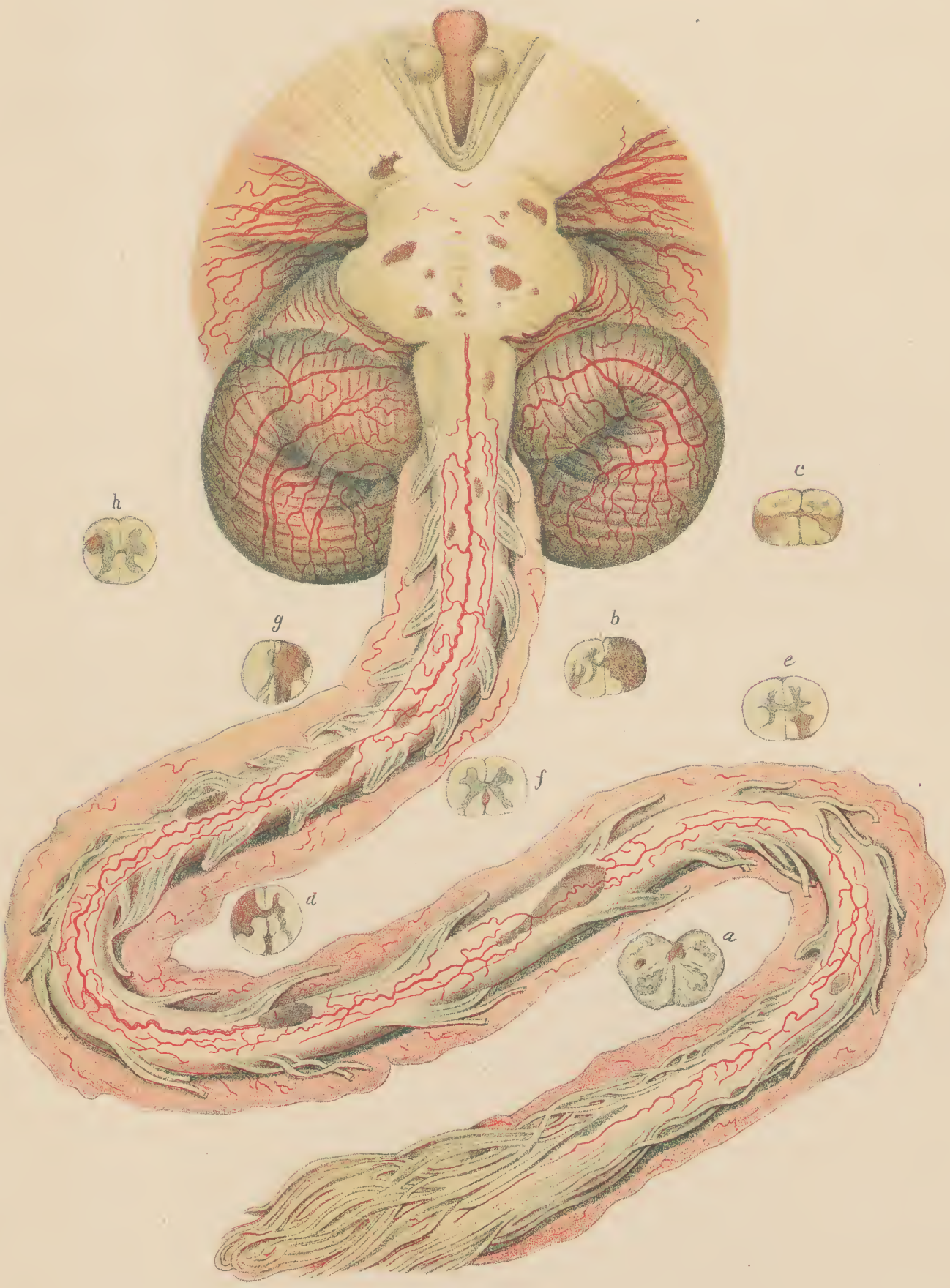


Fig. 3

Fig. 2.

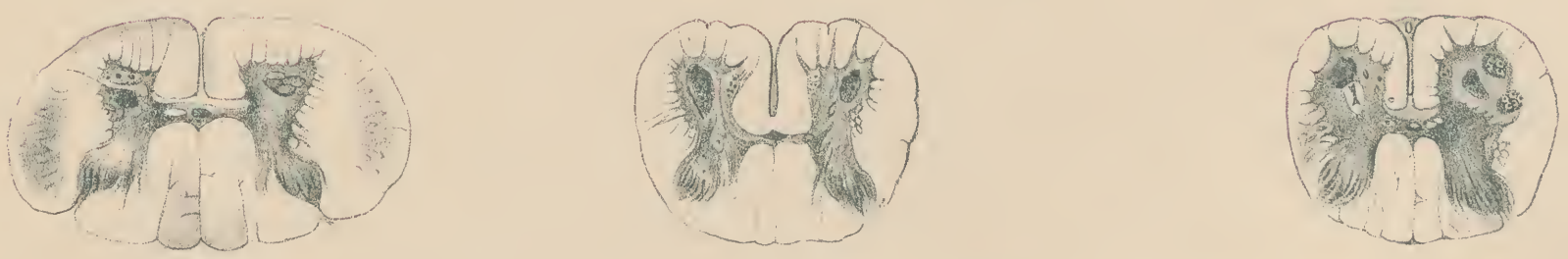


Fig. 4. a.

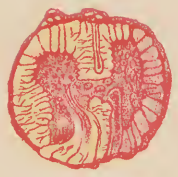
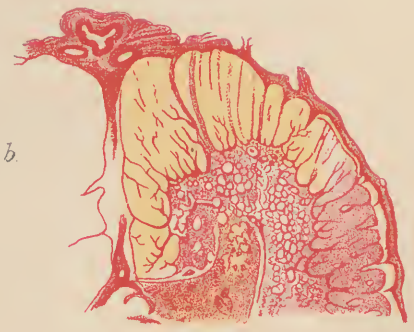


Fig. 4. b.



CATARRHAL GASTRITIS.

ACUTE HAEMORRHAGIC GASTRITIS.

Sec. VI. *Tab. I.*

Fig. 1.

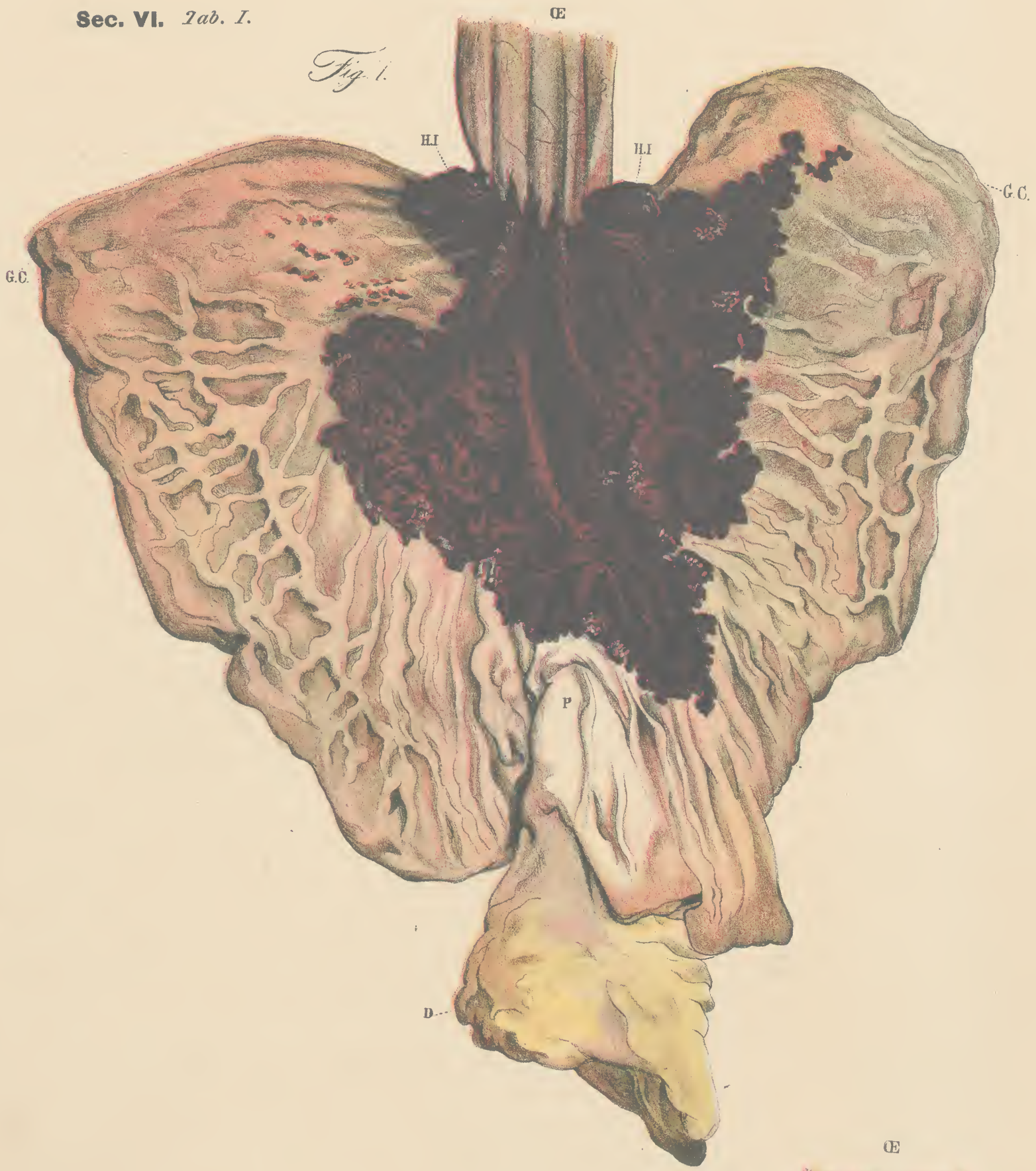


Fig. 2.

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TABLE I.

Fig. 1.—*Catarrhal and Hæmorrhagic Inflammation of the Stomach.*

CASE.—G. L., a four-year-old boy.

History and Symptoms.—On June 7th, 18—, he swallowed a portion of a 14-per-cent. solution of concentrated lye; dropped the vessel containing it and began to scream, and immediately to vomit bloody and mucous masses; was seized with very violent pain in the pharynx, œsophagus, and stomach, which soon spread over the whole abdomen. Repeated retching and vomiting; no stools; all applied remedies did not help. Died after fifteen hours extreme suffering. Post mortem after forty-eight hours. On the chin some corrosion; the lips purple, externally; within, the mouth denuded of its epithelium, and without, spotted reddish brown and greenish. The gums partly spotted and partly opaque white. The tongue, mostly denuded of its mucous covering, intensely red and swollen; the soft palate and pharynx of similar appearance. The swelling, corrosion, and œdematous infiltration extended into the upper orifice of larynx and the sinus pyroformis. The œsophagus partly corroded, partly indurated, discolored, and infiltrated. The peritoneal covering of the stomach pale, and the sub-serous veins very turgid, with black liquid blood. Within the cavity of the stomach, a dark viscid mass, tinged with bile, and of an acid reaction. On its inner surface the cardia, the lesser curvature, and a great part of the adjacent tissue strongly contrasted in color and density against the balance of the mucous tissue. Those formed a triangular elevation above the level of the general surface, distinctly outlined and of brown reddish color, and of consistence of leather. The surrounding surface is red, passing into yellow, with a greenish shade. It is in catarrhal condition. A number of dark red spots are perceptible here and there. A section through the dark red tissue of the triangle shows the whole thickness of the mucous membrane to be involved, the sub-mucous and muscular layers similarly affected. In the lighter portion only the mucous tissue is much affected, with infiltration of serum and lymphoid bodies. The smaller dark spots are injured only superficially. The deeper layers of the stomach contained but very little blood. The greenish discoloration of some portions of the surface was due to post-mortem change of the tissue.

Fig. 2.—*Peracute Gastritis.*

A case of very severe gastro-enteric catarrh, with cholera-like diarrhœa, vomiting, and excessive pain in the whole intestinal tract. Dies shortly after attack. Autopsy very soon after death: Stomach of normal size; innumerable spots of deep red color are spread over the whole inner surface. They constitute miliary hæmorrhages. The color of the membrane proper was yellow, and laid in many longitudinal folds. (Œ) œsophagus, (P) pylorus, (D) duodenum, (G C) great curvature, (H I) hæmorrhagic infiltration.

Pathological Anatomy.

The mucous membranes cover all the inner surfaces of those internal organs which communicate with the exterior of the body. Their structures, although essentially identical with those of the skin, are so far modified in their arrangements as to fit them for the peculiar functions to be carried on in those organs. The sub-mucous tissue is far more loosely constructed than the mucous membrane, and contains far more cells than the other. In some localities it is changed into a lymph-adenoid structure; contains innumerable lymphoid cells, and is covered by a very delicate cover of epithelial tissue. In others the mucous tissue consists of one or more layers of protoplastic cylindrical or columnar cells, and is devoid of any covering of any protective pavement cell layers. All mucous membranes contain abundant quantities of blood and lymph vessels, which are in close contact with the epithelium. From the underlying tissue they are separated by a loosely-constructed connective tissue, which also is plentifully supplied with blood and lymph vessels. In such organs as the stomach and intestines, which are subject to very sudden alternate contraction and dilatation, the mucous membrane is alternately stretched and thrown into folds, and the sub-mucous tissue is very thick and yielding. The epithelium covering the mucous membrane of the intestinal canal is very permeable, allowing liquids and small solid particles to pass from without inwardly and reversely from the blood upon the free surface of the membranes. All mucous membranes produce secretions, made up partly of liquid and partly of mucus, which are formed within the epithelial cells and exudated externally. Great quantities of lymphoid elements also pass from the cellular connective tissue, between the epithelial cells, upon the mucous surfaces, and constitute the spheroid mucous corpuscles. According to Stohr they are most abundant where the mucous tissue contains lymph-adenoid structures. The secretion of mucus is most important to the membrane, not only in physiological conditions, but also, and especially, in pathological, for it constitutes in the latter states a protection against many injurious substances by preventing their immediate contact with the membraneous wall of the organs. Occasionally the lymphoid elements take the place of the mucous in preventing injurious effects upon the organs. When both means of protection prove insufficient or inadequate, then injurious effects upon the membranes will produce more or less change in their structures as well as in their function

Hyperæmia and Hæmorrhage.

The intestinal mucous membranes are subject to periodical hyperæmia necessary to a performance of their function of digestion and absorption. The hyperæmia is brought about by the specific action of the vaso-motor nerves, which permits a greater influx of blood into the tissue than usual. Analogous to physiological hyperæmia are those congestive conditions of the mucous membranes in morbid circumstances, such as paralysis of the vaso-contractors or irritation of the vaso-dilators, which may happen either in the center of those nerves or within the organs. In the hyperæmic state the membrane becomes intensely red, and on close inspection small vessels are found exceedingly turgid. When congestion takes place even the naked eye can discover the hypersecretion which then takes place upon its surface. Static hyperæmia is characterized by a livid color of the membrane. Continued stagnation of the blood usually leads to permanent dilatation of the veins and formation of varices. When œdema follows stagnation the loose sub-mucous tissue can become enormously infiltrated and bloated.

Hæmorrhages may take place in the mucous membrane either to a very limited extent, which will then be manifested by a slight admixture of blood to the secretion or as massive blood infiltration within the tissue and on its free surface. Mechanical injuries, thrombosis and embolisms of the vessels, and certain acrid and poisonous substances are often causes of hæmorrhages; the same takes place in very acute states of inflammation of the membranes. (Section VI, Table I, Figs. 1, 2.) Slight hæmorrhages are, as a rule, soon absorbed, but leave behind them grayish or bluish-black discolorations upon the surface. Ulceration and mortification are not so seldom caused by extensive hæmorrhages. Extensive destruction of the tissues, with total loss of function, often follows such hæmorrhages.

Degeneration, Atrophy and Hypertrophy of Mucous Membranes.

Retrogressive metamorphoses may all find place in the intestinal mucous membranes. In consequence of mechanical, chemical, or inflammatory irritations and derangements of circulation, the membrane may undergo a number of changes in all its structures, changes differing in quality, quantity, and consequences. Alterations in the epithelium are far easier and much sooner noticeable than those of the sub-mucous structures. Catarrhal inflammations show an enormous increase of secretion from the affected epithelial cells. In deep and extensive inflammatory conditions of the mucous membrane extensive exfoliation of the epithelial cells are readily noticeable. Fatty degeneration of that tissue goes on far slower, and never becomes so extensive as the other alterations. The sub-mucous tissue is very often the seat of amyloid changes. The vessels are most frequently thus affected. Atrophy of the mucous tissue and permanent loss of the epithelial covering are very often the result of severe catarrhal affections of the intestinal canal. When the connective tissue between the krypts of Lieberkuhn become decayed from the pressure produced by enormous cellular infiltration, and the epithelial lining of those krypts loosen and exfoliate, the glandular layer of the membrane becomes atrophic and its glands obliterated. This atrophy superinduces atrophy of the sub-mucous muscular tissue. However, when the lesion is not too deep and the epithelial loss is not too extensive, the whole surface is very liable to be speedily regenerated. At other times there are produced granulations and hyperplasia, with more or less cicatricial contractions of the membrane. Should neither take place there will remain a permanent sub-acute ulceration. Hyperplastic formations are not uncommon in the intestinal tract, and may assume a variety of forms to be described hereafter.

The Stomach.

The stomach is that part of the intestinal tract where the process of digestion and absorption begins. Accordingly it is provided with great numbers of blood, lymph vessels, and very numerous glands, which secrete gastric juice and mucus. As digestion requires a long stay of the food in the cavity of the organ, and at the same time an increase of the quantum of blood in its walls is produced, which causes production of its secretions far beyond that found in an empty stomach, its epithelial tissue undergoes a certain amount of disintegration. Both conditions, although physiological, are very much akin to those morbid processes which are usually observed in inflammation. The massive proportion of the sub-mucous structures, its great quantity of muscular tissue and large amount of labor all its tissues have to perform during stomach digestion, expose this organ to a greater number of casualties than any other organ of the body, except the skin. On the other hand, the stomach is provided with enormous power of resistance to injuries and an equally great power of regeneration and reconstruction of its injured or destroyed tissues.

Many substances, which, in their passage through the mouth, pharynx, and œsophagus, into the stomach, affect very seriously the walls of those passages, affect but little those of the stomach. This is due to the power of this organ to protect itself against many injurious effects by secreting large quantities of mucus, which, in a

TABLE II.

FIG. 1.—*Acute Catarrhal Gastritis with Infiltration into and Hyperplasia of the Mucous Membrane.*

The stomach is here slightly dilated, the mucous membrane thickened, and its folds very prominent. The openings of Lieberkuhn's glands are enlarged and surrounded by circular ridges, plainly showing the orifices of the dilated sacs. The follicles are increased both in size and number, especially in the pyloric and œsophageal regions. The appearance of the membrane is less red than that shown in Table I, Fig. 2, of this Section, yet sufficiently injected to show a high state of hyperæmia and severe catarrhal inflammation.

FIG. 2.—*Acute Gastric Catarrh, Corrosive Ulceration of the Mucous Membrane of the Stomach, Œsophagus and Pharynx. Laryngeal Croup.*

CASE.— A child six years old.

History.— Is supposed to have swallowed some corrosive liquid. Antidotes and other proper remedies to counteract the supposed poison were administered. The child constantly complained of pain in the throat and in the œsophagus, without being relieved by the treatment. Swallowing became more and more difficult, dyspnœa appeared on the fourth day and gradually increased in intensity to the twelfth day, when it died of asphyxia. Post-mortem appearance after three days: The slightly swollen lips are brownish red, tough, and covered with thick, dark-red crusts. The inner surface of the lips and the gums are opaque white, and unimpaired. The soft palate, pharynx, œsophagus, and the region of the cardia are corroded in many places and present opaque white and yellow spots. In some places the surface-tissue still adheres to the underlying, while in others there is solution of continuity between them. The injuries differ in the upper part of the œsophagus from the lower in depth and intensity. The lesions of the stomach are not very extensive. Besides those situated in the cardia there are in the left half of the posterior wall and nearly in the middle of the organ a moderate number of ulcers. The abraded portions are surrounded by wider or narrower hyperæmic borders. The swelling, even in the vicinity of the extensive ulcers, is not very great. The edges of the largest ulcers are abrupt on nearly all sides. The bases of the smaller ulcers are in the middle layer of the mucous membrane, while the larger reach into the sub-mucous structures. A number of small, shallow ulcers are scattered over different portions of the inner surface. The pale-red mucous membrane shows a state of catarrhal inflammation, and is covered in many places with minute hæmorrhages of very recent date. The membrane is covered with a tough, glassy mucus, slightly tinged with bile. The deeper layers of the walls of the stomach are unaffected.

great measure, prevent immediate contact of the injurious substances with the walls of the organ. But this power of hypersecretion has its limits; and when the injury is continuous, the stomach becomes so much deeper affected, for the epithelial tissue becomes the less able to resist the more the exhaustive supersecretion has taken place. By far the commonest affection of the stomach is catarrhal inflammation, which may either be acute or chronic. In acute catarrh the mucous membrane is tinged either all over or in spots, dark red, swollen, and spread over with hæmorrhagic spots of various dimensions. (Section VI, Table I, Figs. 1, 2.) The surface is usually covered with mucus, degenerated epithelium, and lymphoid cells. The cylindrical epithelium of the glandular orifices is in a state of mucous degeneration and exfoliation; the epithelial cells of the peptic glands are displaced and more granular than usual. The interglandular connective tissue contains very turgid blood vessels, and is infiltrated with lymphoid cells, more especially along the veins. The sub-glandular and sub-mucous structures are also much infiltrated, and the cells in the lymphatics are deeply altered. Such inflammatory conditions may exist within the whole stomach or only in parts. The pyloric region is usually affected in this manner. By far the greatest number of cases of gastritis are readily cured and very transitory, but here and there it assumes a chronic form which leads to extensive and deep changes in the organ.

When the epithelial covering undergoes extensive destruction from massive infiltration, etc., it is hardly ever regenerated, but is followed by an atrophic condition of the glandular parenchyma. When the infiltration is excessively great, then both epithelium and connective tissue become destroyed and utterly obliterated. More or less extensive *ulceration* is then produced, which gradually diffuses over the whole surface. The ulcers differ in size, their depth is unequal, their limits indefinite. On the bottom of such ulcers numerous elevations of various heights, and differing in color, may be found. The edges of the ulcers either pass imperceptibly into the adjacent portions of the healthy tissue, or are distinctly or indistinctly outlined, and covered with excrescences or vegetations in the form of polypi or ridges. When the ulcers increase to a size readily visible to the naked eye (Section VI, Tables II, III, IV), the glandular structures are found perfectly obliterated, and the muscular tissue, although still preserved, is filled with lymph corpuscles. The sub-mucous tissue is indurated and thickened, and contains far more connective-tissue cells and lymphoid corpuscles than the normal. Preserved portions of the mucous membrane are also infiltrated, especially in the neighborhood of the indurated parts. Some of the glands are very much constricted, some enormously dilated, and the lymph follicles more numerous and much larger than normal. (Section VI, Table II, Fig. 1, F F.) Such extensive ulcerations after inflammation are rather of rare occurrence, but still sometimes do take place. Pigmentation, atrophy of the glandular portion and partial induration very frequently follow inflammation of the gastric mucous membrane. The pigment is here as everywhere the remainder of former hæmorrhages, and presents grayish or gray-black spots. The atrophy of the membrane is easily recognizable by its thinness. When hyperplastic formation is carried on to a very moderate extent, only the interglandular structure will be found increased in density, but when it takes place in a massive manner then the mucous membranes become sclerotic, and elevations in the shape of ridges and folds will appear upon parts or nearly the whole inner surface of the organ. (Section VI, Tab. VI, Fig. 4.) This rugous or warty appearance is designated by French writers as "*Etat mamelne*," *or gastric polyposis*. The hyperplastic tissue is sometimes tough and fibrous, and sometimes soft and cellular, and owing to loss of the epithelium part of the mucous glands becomes obliterated and part extremely dilated. The dilated cysts contain muco-serous liquid, crumbling epithelial detritus, and often colloid balls. Larger cysts are sometimes filled with papilliform excrescences. In all chronic forms of gastritis, and especially in the ulcerative, the development of connective tissue extends into the sub-mucous, the muscular, and even serous coats, and renders the walls of the stomach dense and indurated. The muscular tissue is sometimes completely obliterated, or becomes hypertrophic.

Phlegmonous inflammation of the stomach takes place either locally or generally; the latter is the most common. The seat of the inflammation is essentially in the sub-mucous tissue. In the circumscribed form perforating abscesses are formed. In the diffuse form the inflammatory process begins in the sub-mucous tissue by swelling and rendering it more dense, whilst the mucous tissue becomes either moderately swollen or remains unaltered The exudate is of a viscid puriform nature and often infiltrates into the muscular coat, extending sometimes even into the serous. Both become swollen, and the serous surface is often covered with pus or fibroid-pus. In the advanced stages the tissues become softened, and the pus perforates the mucous membrane here and there; the muscular coat may likewise become perforated. Should the patient survive (which sometimes happens in small circumscribed phlegmons, and provided there is no perforation into the peritoneal cavity with discharge of pus), cicatrization will close up smaller perforations, and larger ones become covered with epithelium but permanently lose the sub-mucous structure.

Idiopathic croupous and diphtheritic inflammations of the stomach are very rare, but as a part affection of pharyngeal and œsophageal croup or diphtheria is of common occurrence. In scarlet fever, small-pox, and in other very acute infections, croupous exudate, more or less extensive, is found upon the mucous surface either in the form of yellowish-gray membranes or plaques. In diphtheritis the superficial layer of the membrane becomes escharotic, and the gangrenous process may extend into sub-mucous tissue and convert the whole into a black mortified mass.

Hæmorrhages.

The great vascularity of the stomach makes it readily subject to hæmorrhages. Mechanical, chemical, thermic injuries, inflammatory and other morbid alterations of the vascular walls, ulcerative processes, venous stasis in the liver and portal system, scurvy, yellow fever, acute yellow atrophy of the liver, typhoid and severe exanthematous fevers, etc., local as well as general infections and various dyscrases may produce gastric hæmorrhages. Extravasated blood upon the inner surface of the organ soon assumes a deep brown or black color, owing to the reduction of its hæmoglobin to hæmatin by the acid secretion of the stomach.

Hæmorrhage into the mucous membrane makes the tissue look either red, brown, or grayish black. Circulation in such an infiltrated tissue is either carried on very incompletely or not at all, and consequently becomes subject to digestive action of the gastric juice and is soon destroyed. (*Autopepsia.*) When the tissue is destroyed more or less extensive bleeding ulcers are formed. (Hæmorrhagic erosions.) Provided the necessary precaution is used by the affected person such ulcers very readily heal. Regeneration of the destroyed tissue begins with reactive inflammation, infiltration of the tissues adjacent to the ulcers, followed by formation of granulations and connective tissue; finally reproduction of an epithelial covering.

Pathology.

Morbid alterations of the blood vessels of the stomach, which either hinder or check altogether local circulation in the organ, are the main causes of corrosive action of the gastric juice upon the anæmic tissue. Virchow, Rokitansky, Merkel, and others have proved that recently-formed ulcers were due to closure of the cavities of the smaller gastric arteries, brought about by embolism, thrombosis, fatty degeneracy, atheromatous or amyloid alteration of the walls. Pavy has shown that by ligating the gastric arteries of animals ulceration in the stomach is produced. In fact any cause capable of arresting gastric circulation to any great extent will produce circular ulcers.

DISEASES OF THE ORGANS OF DIGESTION.

GASTRITIS WITH EXUDATION AND HYPERPLASIA.

PARTIAL EROSION.

Fig. 1.

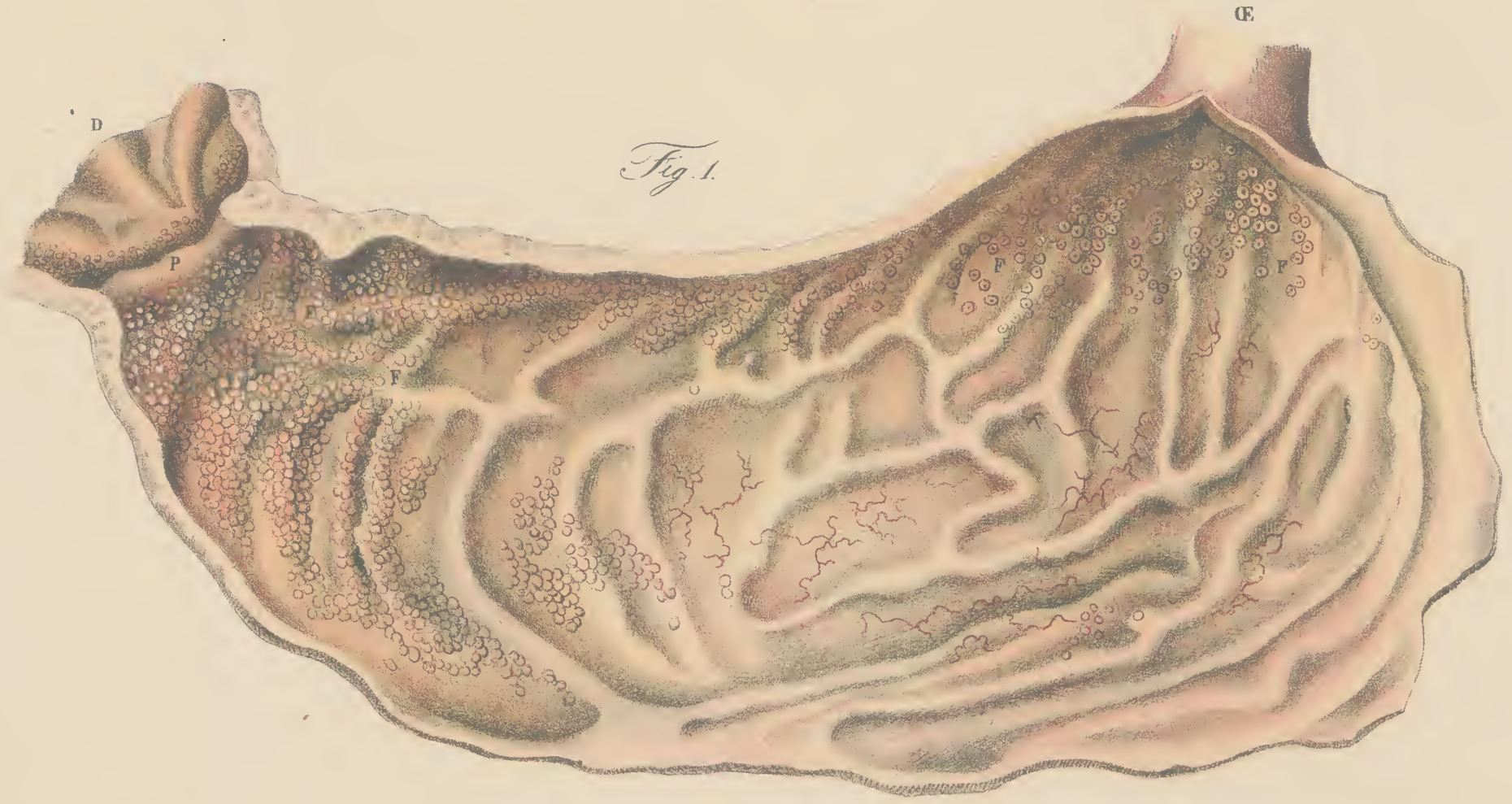
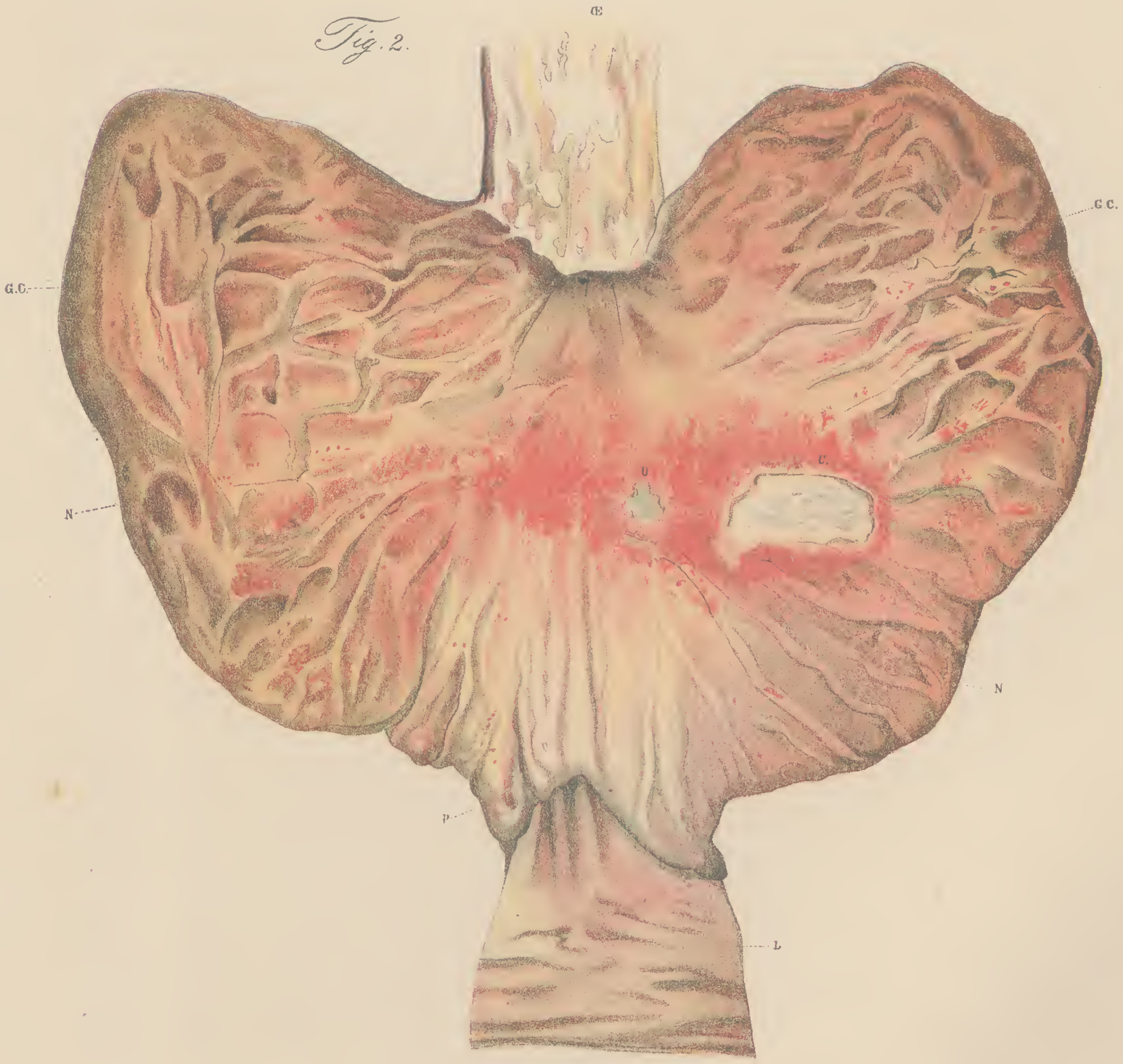


Fig. 2.



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HAEMORRHAGES AND HAEMORRHAGIC ULCERATION.

Sec. VI. *Tab. III.*

HAEMATIC INFILTRATION.

Fig. 1.

Fig. 2

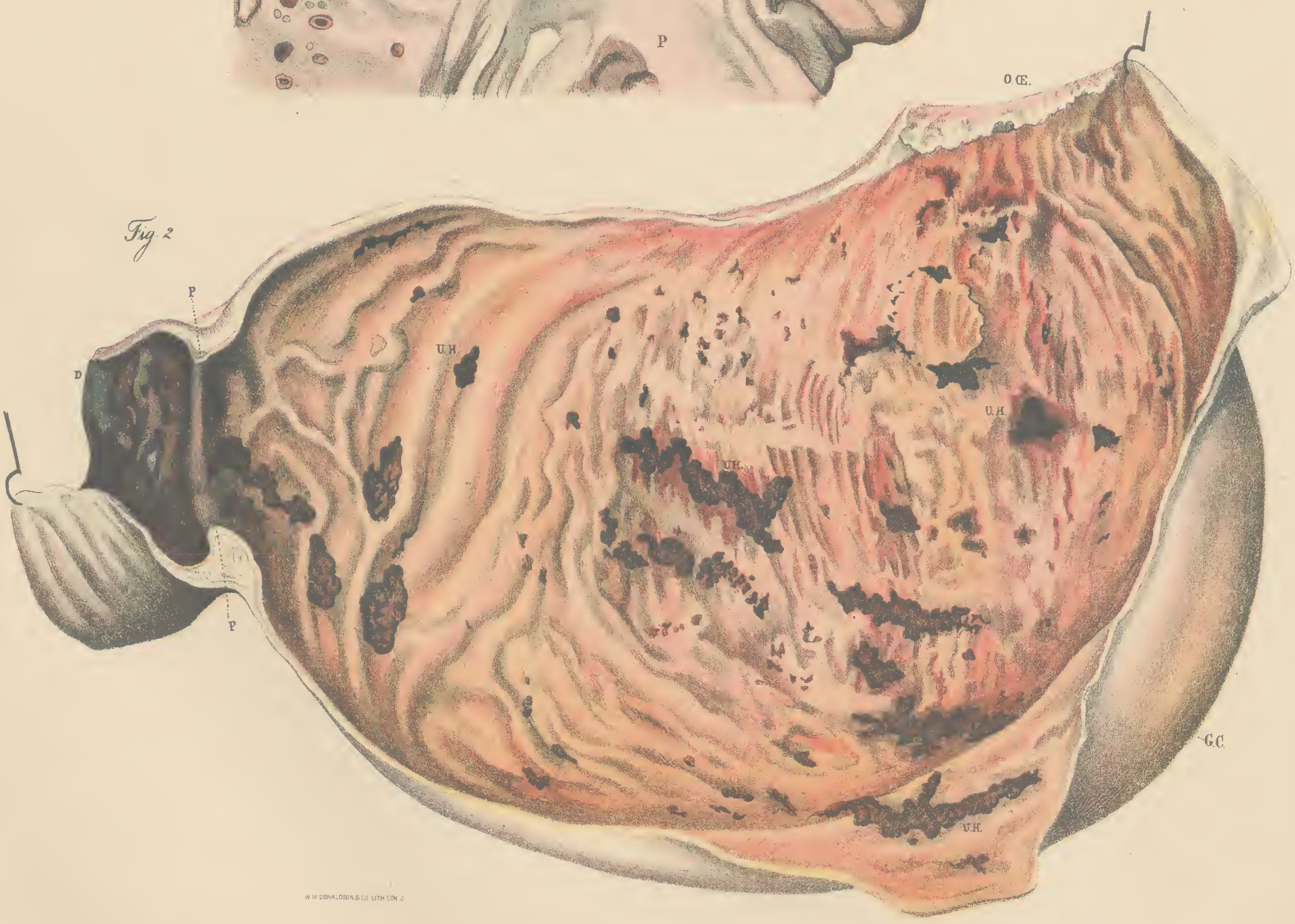

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TABLE III.

FIG. I.— *Hæmorrhages and Hæmorrhagic Infiltration into the Mucous Membrane of the Stomach, Œsophagus, and Duodenum.*

CASE.— A woman forty-seven years old.

History and Symptoms.—Was treated several weeks for syphilitic ostitis and chronic amyloid degeneration of the parenchyma of the kidneys; a very rapidly extending erysipelas from very extensive bed-sores on the sacrum supervened, with imminent collapse. In spite of all treatment, debility increased, and finally she was carried off by uræmia, from insufficient action of the kidneys.

Post-mortem appearance: The cavities of the mouth, pharynx, and upper portion of the œsophagus were free from recent morbid changes. Cicatrices of old syphilitic sores visible on the soft palate and the root of the tongue. The effect of the benzoic acid and camphor in powder with which she was treated showed itself in various ways upon the mucous membrane of the stomach. In some parts there were simple hæmorrhages (especially in depressions between the folds) into the membrane. In some there were erosions; their edges and fundus were infiltrated, some were blackish brown, some light-colored, some free from any infiltrate. They were mainly of a circular or oval form, and of different dimensions (E, E, H, U). They were shallow and within the mucous membrane proper. The ulcers produced here by hæmorrhagic infiltration were similar to those caused by corrosive poisons, for here the glands and interglandular tissue were deprived of the usual protection of the membrane, that is, the alkaline serum, against the corroding effect of the acid gastric juice, as the extravasates checked all circulation in the membraneous wall. In some places the destructive process went on slower than in others, and correspondingly the color was lighter or darker. In some parts the membrane had a bluish or greenish tinge, showing a more or less advanced stage of post-mortem putrefaction. The black spots were produced by the reducing agency of the acid in the cavity upon the hæmoglobin of the exudate. In the left half of the cavity the membrane had lost its opacity, became transparent, and showed through the turgid vessels, the color of which varied according to the amount of decomposition which they had already undergone.

FIG. 2.—This represents a form of hæmorrhagic erosion of irregular form, some being situated very deep in the gastric walls, some very superficial, thereby indicating the progress of the destructive process. (G C), great curvature; (H U), hæmorrhagic ulcerations; (E E), erosions; (O E), œsophagus; (P), pylor; (D), duodenum; (H I), hæmatic injection or infiltration.

Ætiology.

According to B. Ziemssen (Clinical Lecture, No. 15, Volkmann's Collection), the number of females affected with simple circular ulcer is double that of males. The statistics gathered by Brinton show that the frequency of the disease increases from the age of ten years and upward in both sexes. Chlorosis and anæmia especially predispose young persons to become thus affected, as Virchow and Rokitansky have both proven that the thin walls of the arteries of chlorotic young women readily undergo fatty degeneration. Such friable conditions of small arteries and capillaries will, in many instances, even under the slightest provocation, break and produce hæmorrhages and hæmorrhagic infiltration, and coincidently lack of circulation in the mucous membrane of the stomach and subsequent ulceration. Complication of gastric ulceration with tuberculosis and chronic pneumonia is a very common occurrence. Inadequate nutrition of the bodily tissues is, according to Bamberger, the probable cause of both processes.

Hæmophilia, produced by chronic endocarditis and affections of the internal membranes of the blood-vessels, also, according to Siebert, derangements of vascular innervation are often the predisposing causes of gastric ulceration. Formation of ulcers takes place in the stomach and the duodenum, and in very rare cases in the lower portions of the œsophagus. When nutrition of any portion of the mucous membrane has ceased for any length of time, there is formed, very speedily, softening of the hæmorrhagically infiltrated portion. A distinctly circumscribed circular portion of the membrane turns into a sphacelous mass or a dry scab, and these become detached from the sub-mucous tissue. This eventually undergoes the same disintegration; sometimes even the muscular and serous coats become involved in this manner and are destroyed. Merkel describes a case of a ninety-four-year-old woman in whose stomach there was found, post mortem, a circular ulcer, in which the mucous membrane still existed, but was changed into a black mass and adherent to the fundus of the ulcer. Sometimes the membrane is perfectly obliterated and the wound is funnel-shaped, the base turned outward, its apex within the deeper coats. The funnel shape seems due to greater and more speedy destruction of the mucous membrane by corrosive action of the gastric juice than the tissues of the deeper layers. In older wounds the sub-mucous and muscular layers become more disintegrated, and the loss of substance becomes more uniform throughout. The extent of chronic ulcers is always greater than of those of recent date. Rindfleisch, in reporting a case of acute circular ulcer having the above-described form, builds his theory of the formation of such lesions upon the fact that they are always found when any hæmorrhagic infarct extends through the whole thickness of the membrane. "When such infarcts become dissolved by the gastric juice," he says, "then circular wounds are formed in the mucous membrane, and are of such regularity and have the appearance as if a portion of the membrane had been cut out by a circular punch, leaving the surrounding and underlying tissue perfectly clean. Such infarcts take place within the limit of a definite territory of circulation of an artery in the walls of the stomach, and these all have a conical shape, their base facing the epithelial surface, their apex turned inward." When the ulcerative process reaches deeper into the sub-mucous structures the same circular form is maintained for quite a long while. "It seems," says the same author, "as if the formative reaction at the fundus and the sides is so slight, and especially as the action of the gastric juice follows so speedily the plastic reaction, that there is but little possibility of formation of any hyperplastic infiltration in any parts adjacent to the ulcer." "The fact that ulceration may continue a great number of years in the same simple condition, and also that they are only found in the stomach and in the upper portion of the duodenum, where the secretion is of an acid reaction, and consequently capable of dissolving liquid or liquefied albumenates, go very far to explain the peculiar phenomena of the simple ulcer." (Rindfleisch.) The smaller the ulcer, the more recent the destruction of the tissues, the oftener and sooner will there be formed cicatricial restitution. As a general thing the cicatrices involve a far greater portion of the gastric wall than the destroyed tissue (Section VI, Table V, Figs. 1, 2, 3). Where cicatrization follows very extensive ulceration (which happens very seldom), there are produced constrictions of the cavity of the stomach, which become a source of annoyance and suffering to the patient. The dangers liable to beset the ulcerative process are: first, very profuse and often fatal hæmorrhages, caused by corrosion of smaller or larger blood-vessels; second, anæmia following often-repeated profuse hæmorrhages; third, perforation into the peritoneal cavity. This may happen before any attachment of the stomach to any neighboring tissue has been formed, or even afterward, for the adhesive structure readily becomes detached, and the contents of the stomach are then emptied into the peritoneal cavity, which always terminates in fatal peritonitis.

When the ulceration is followed by hyperplastic structure, forming adhesion of the organ to any other, the adherent surface is always increased in thickness and density. Yet such cicatricial tissue is sometimes very friable and readily allows breaking through of the gastric walls and emptying its contents into the parenchyma of the supporting organ. Under such circumstances, formation of abscesses and pyogenic sinuses will result. Such adhesions to and perforations into may befall almost any organ within the abdominal and thoracic cavities. (*Virchow's Archiv., Vol. V. Klebs, Handb. d. Patholog. Anat., Vol. I. Ziemssen, Collect. Clinic. Lectures, No.* 15. *Gerhard, Wien. Med. Press,* 1868. *L. Muller, Corros. Ulc., Erlang.,* 1880. *Boettcher, Dorpat. Med. Zeit.,* 1874. *Rindfleisch, Pathol. Anat. Ziegler, Pathol. Anat., Part II, p.* 305.)

Tumors and Other Neoplasmata of the Stomach.

The most frequent, also the most important, heteroplastic formations of the stomach are the many varieties of cancer. It always first takes its origin in the mucous membrane, and from there very soon passes into the sub-mucous tissue, where it often extends very rapidly, and then involves the muscular and sometimes even the serous coat of the organ. In the serous covering it usually assumes the nodular form, and follows the tract of the lymphatics (Section VI, Table VII, Figs. 3, 4). These nodules or nodes often invade the cavities of the veins and produce cancerous thrombi, appearing upon the serous surface, especially in the pyloric region, as flat or knotty strings (Section VI, Table VIII, Figs. 4, 5). Early in the course of the disease, the lymphatic glands situated in the region of the lesser curvature become enormously enlarged and form massive cancerous knots (Section VI, Table VIII, Fig. 2). Occasionally metastatic deposits of cancer will be produced in the liver, lungs, etc. The portal vein is usually the most frequent way of propagation of such cancerous metastases of the liver. The most frequent form of cancer of the stomach is that of raised, soft, fungoid tumors in the pyloric region (Section VI, Table VII, Figs. 1, 4, 5), also in the lesser curvature. They are very rare in the large *cul de sac*, rarely also do they spread over the inner surface of the organ. When the tumor has reached a certain size its central portion soon undergoes a process of decay, changes into a cancerous ulcer, characterized by being surrounded by raised edges. The fundus of such an ulcer is formed by the sub-mucous tissue, which is either infiltrated with cancerous cells or becomes indurated in consequence of chronic inflammation (Section VI, Table VII, Fig. 2). Usually the muscular as well as the serous coats are extensively hyperplastic, enlarged, and indurated. Sometimes the whole pyloric region is thus infiltrated, enlarged, and sclerotic. At other times the whole neoplasm on the inner surface of the stomach becomes so decayed and atrophic as to render the whole surface smooth and having the appearance of a fibroid, with no malignant induration. Even the microscope will fail to discover any cancer cells in such a tissue, and only the metastatic deposits found in the co-ordinate digestive organs will reveal the existence of cancer.

According to the histology of each, Cancer can be divided into five divisions:

1. *Medullary Cancer*, consisting of soft, spongy tumors, producing either ridge-like, nearly parallel elevations upon the inner

TABLE IV.

FIG. 1.— *Acute Hæmorrhagic Gastritis, Primary Degeneration of the Parenchyma, Multiple Embolic Foci, Exudate upon the Surface.*

CASE.—A girl twelve years old.

History and Symptoms.—She was taken with pharyngeal diphtheritis about two weeks before her death. On the tenth day of her sickness she vomited blood copiously and had bloody stools. The defecations continued very frequent until her death. On the day of her death she had very severe nose-bleeding. Died from exhaustion. The body was examined the third day after death and showed post-lethal dark spots in many places. Hæmorrhages in all serous membranes and cavities. Great alteration in myocardium and kidneys. Spleen normal, soft palate, tonsils, uvula, and adjacent tissues, the root of the tongue very much swollen. Their superficial structures opaque, dirty gray color, in places green, and reduced to a soft pulp, consisting of debris of the tissue and enormous numbers of different-shaped *cocci* and bacteria. The deeper tissues, in consequence of profuse infiltrations, are deep red, and contain many congeries of *micrococci*. All tissues of the larynx, the lymph-glands of the neck, etc., in a high state of œdematous infiltration, some hyperæmic, some anæmic.

In the considerably dilated stomach, there is a blackish, soft, pulpy mass. The mucous membrane is covered all over with a layer of thick mucus, which has preserved its original glossy appearance. In the pyloric region and in some other localities the mucus is stained by an admixture of blood. The greatest portion of the mucus is here represented as removed, and shows the surface of the cavity in a thickened and slightly turbid condition. A great many hæmorrhagic points contrast with the slightly anæmic general surface. The color and extent of the foci differ very much. Some are circular, some oval, some have a very bright red color, some a deep cherry red, some are perfectly black. Depth of color is in ratio to the amount of reduction of hæmoglobin.

It seems the gastric juice has not acted everywhere alike in extent and duration. The yellowish-red portions of the hæmorrhages are due to a combination of bloody and cellular infiltration. They were produced by irritant emboli, which turned into hæmorrhages. Some of these contain micrococci, some do not, but all showed a deep disturbance of nutrition. The contents of the intestines were also mingled with blood, evidently derived from the stomach.

FIG. 2.— *Scarlet Fever and Acute Gastritis. Hæmorrhages, Erosions, also Biliary Imbibition in the Walls of the Intestine.*

CASE.— A five-year-old boy.

History and Symptoms.— About two weeks before his death he became unwell, restless, and lost his appetite and his ordinary good humor. Was peevish, and at times, chiefly in the night, feverish. Three days before death his skin became intensely red, and he complained of thirst. Twenty-four hours before death he took some food, and very soon began vomiting and purging, which, with excessive weakness and pain in the head, fever and great thirst, lasted until his death.

Post-mortem examination forty-eight hours after death. The skin, the cranial and thoracic organs offered nothing abnormal. Besides some parenchymatous alteration of the kidneys, only some portions of the intestinal tract were affected. The tonsils and pharynx were swollen and very red; the tongue and œsophagus pale, but not at all affected. The stomach somewhat contracted, its serous covering pale, delicate, and smooth, the sub-serous veins extensively distended with dark, liquid blood. The mucous surface was covered with tough, glossy mucus, intensely stained yellow with bile. Its superficial layers, with few exceptions, are all of a yellow color; the folds are most intensely so. The pyloric region has retained most of its natural hue. In some of its parts there are a few injected vessels. Some spots are pale or stained yellow. (The letters in these figures correspond to those found on the former page.)

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surface of the organ (Section VI, Table VIII, Fig. 1), or single knots. Decay of the central masses produces ulcers with raised edges of a yellowish-white color. This cancer is developed from the gastric glands. Histologically it is characterized by very numerous cancer cells in the little-developed struma. Beneath the decayed portions there is produced fibrous induration and cancerous infiltration in the gastric walls. This form most frequently causes metastatic deposits.

2. *Epithelioma.* It forms soft, knotty tumors, which afterward become ulcerated. It constitutes tubular vegetations (Section VI, Table VII, Figs 4, 5), which are provided with a simple cylindrical lining, very much resembling tubular glands. They very soon lose their glandular appearance. The neoplasm is usually soon destroyed by ulceration, and carcinomatous and connective-tissue infiltration takes place at the bottom of the ulcers.

3. *Scirrhus or Fibroid Carcinoma.* This form is usually formed in the region of the pylor, where the walls become indurated and increase in volume. The pyloric opening thereby becomes more or less contracted. The inner surface of the indurated part is either covered with many hyperplastic folds of the mucous membrane (Section VI, Table VIII, Figs. 4, 5), or the tough and enlarged sub-mucous tissue protrudes through the mucous membrane. A section through the walls still shows them to be separate but very much infiltrated with connective-tissue cells.

4. *Colloid or Gelatiniform Carcinoma.* This presents itself in the form of tuberculous single tumors, or as a diffuse infiltration in the walls of the stomach. In both the neoplasmata contain a jelly-like substance, very transparent and very soft. It is often extended upon the peritoneum, forming very translucent, jelly-like, more or less vascular masses (Section VI, Table VII, Fig. 1). The jelly is derived from both the cancer and connective-tissue cells. Colloid often appears very early in life, while the other described forms affect people in advanced age.

5. *Pavement Epithelial Cancer.* This is the rarest of all forms in the stomach. Its usual seat is in the cardia and its surroundings. Myoma, sarcoma, and fibroma are very seldom formed in that organ.

Functional Disturbances of the Stomach.

Whenever the organ becomes affected by disease, there will invariably ensue more or less functional disturbances, which may also lead to further new derangements, or to additional changes in its tissues.

As the gastric secretions produce definite changes in the food taken, any modification in the structure will necessarily produce abnormal changes upon the contents of the cavity. More especially is this brought about by the presence of *schizomycetes* (fissiparous fungi), which are constantly introduced into the stomach with the food, drink, and other substances. Normally the gastric juice prevents these organisms from becoming active and increasing, but when it becomes vitiated by disease or ceases to be secreted altogether, then those fungi will become active and increase to an enormous extent. More especially is this the case when the stomach becomes distended with food which cannot, under the circumstances, be digested, and remains stagnant in the cavity. A state of insufficiency or inability of the stomach is produced by taking too much food and too frequently. Such insufficiency is usually followed by very disastrous consequences to the patient. Continued pressure upon the stomach by clothes or accoutrements, voluntary or involuntary unnatural position of the body, especially of the thorax and abdomen, tumors in the stomach or in the adjacent organs or parts, all of which are liable to prevent timely removal of the food from the stomach into the other intestine, the same as constriction or contraction of the pyloric orifice of the gastric end of the duodenum, will produce dilatation of the organ. When food is not properly digested in the stomach it remains a long while in its cavity, and the vast variety of micro-organisms will develop all their activity upon the stagnant and decomposing mass and produce a great number of ferment-products, which will increase and further aggravate the diseased condition of the organ.

In a morbidly dilated stomach there is to be found a vast collection of spora and more or less developed organisms, producing lactic, butyric, acetic, and alcoholic fermentations. Their products exert exceedingly injurious effects upon the walls of the stomach, cause hyperæmia, hypersecretion, and often a continued state of congestive irritation, protracting diseases of the organ indefinitely and turning acute catarrhal gastritis into chronic.

Recklinghausen has demonstrated that schizomycetes often penetrate into the Lieberkuhn's crypts, and from those pass into the parenchyma of the organ, producing pustulous enlargements in the mucous membrane. (Virchow's Arch., Vol. XXX.)

Very many cases of mycotic gastritis which lead, sooner or later, to the death of the affected persons, under cholera-like symptoms, have been reported. Post mortem, the stomachs were found ulcerated and within the ulcers an endless number of micro-organisms, thriving upon the decomposing and decomposed substances of the organs.

When the quantity of acid is greatly increased in the contents of the stomach and the circulation in the mucous membrane is diminished, there is formed a state of maceration and softening of the membrane and its self-digestion. According to the quantity of blood in the organ, the destroyed tissue assumes a brown or a black color, and is converted into a pulpy, gelatinous, very friable mass. This constitutes the so-called *gastromalacia.* This form of softening is most frequent in diseases of the brain and tubercular meningitis. Usually this destructive process goes on during death-agony, terminating many infectious and contagious diseases, also when the agony is much protracted in formerly healthy persons. (*Leube, Kussmaul, Jurgensen, Ziegler.*) *Samulson* doubts very much that an increase of acidity in the gastric juice is alone capable of producing gastric self-digestion; also that alkalinity of the blood is the natural protection against the corrosive action of that secretion upon the mucous membrane. He cites a number of experiments he made upon animals by producing extreme acidity of the gastric juice, in a variety of ways, and in which no malacia was produced. He considers that there must be a peculiar disturbance in circulation to produce that softening, for he had severed the gastric filaments of the Vagus nerve after rendering the gastric secretion very acid, and found no malacia following it in the stomach of the animals thus injured. He considers that the cause of gastromalacia during life has yet to be learned. (*Selbstverdauung. Inaug. Dissert. Jena.*)

DISEASES OF THE ORGANS OF DIGESTION.

PRIMARY DEGENERATION OF THE TISSUES.
MULTIPLE EMBOLI.

EXUDATE UPON THE SURFACE OF THE MUCOUS MEMBRANE.

Sec. VI. *Tab. IV.*

Fig. 1.

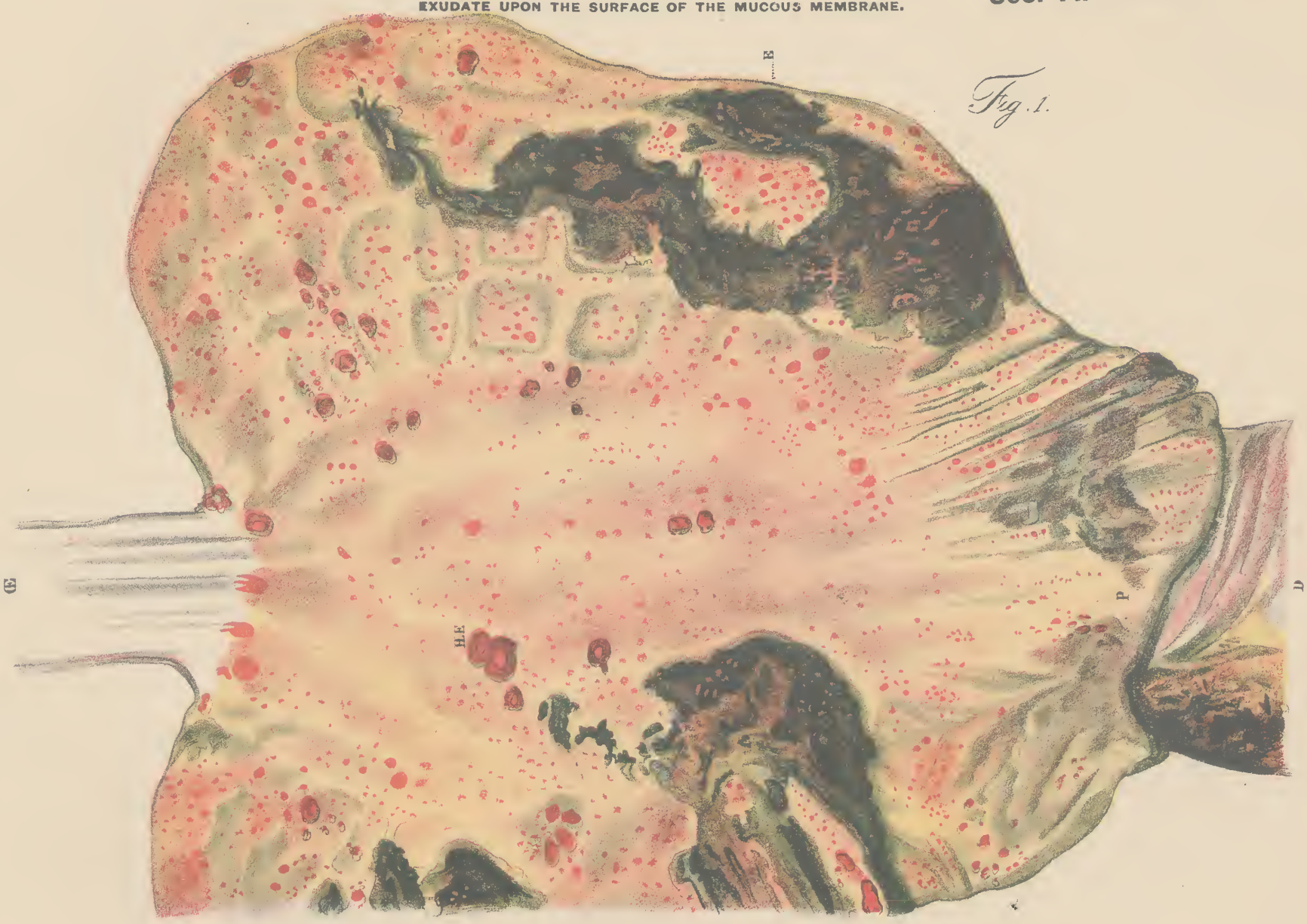
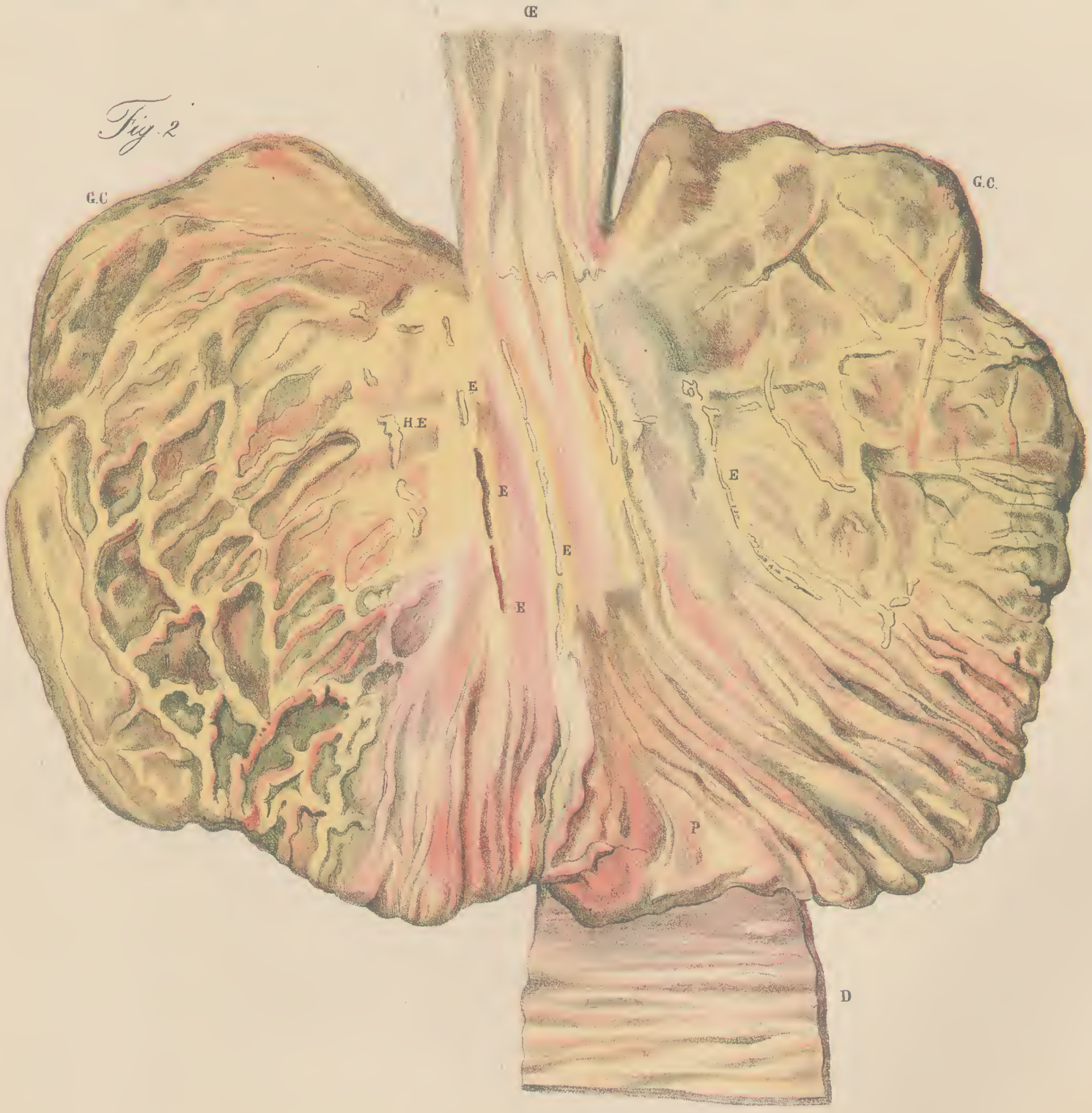


Fig. 2



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Sec. VI. *Tab. V.*

IDIOPATHIC PHLEGMONOUS GASTRITIS.
SOFTENING AND PERFORATION.

Fig 2

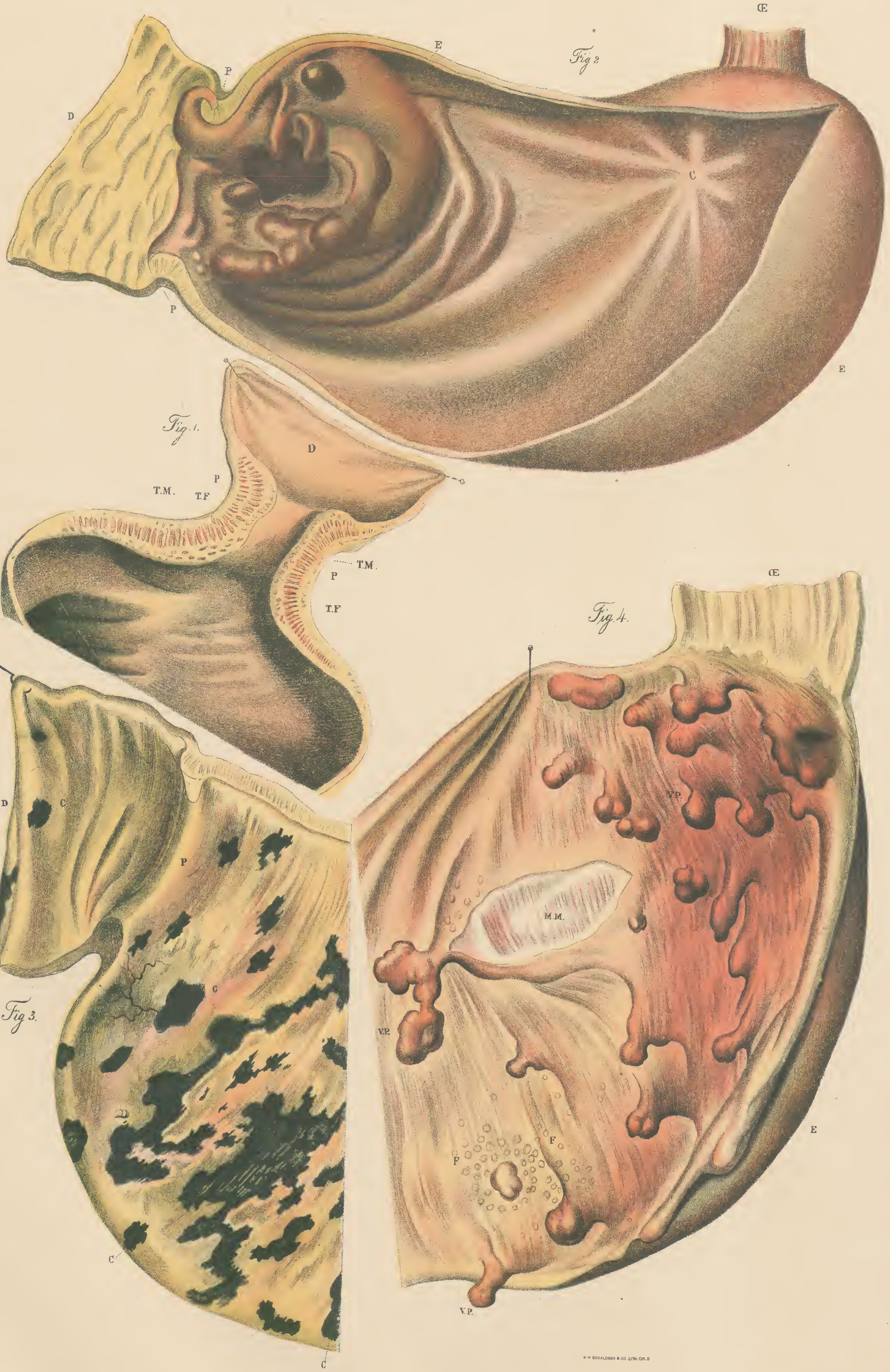

Fig. 1.

Fig. 4.

Fig 3.

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TABLE V.

FIG. 1.—*Idiopathic Phlegmonous Gastritis.*

CASE.—A robust man of fifty.

History and Symptoms.—After returning from a trip in the country with his family, where he partook of ordinary food, he was suddenly seized with violent pains in the stomach and vomiting. Pulse moderately frequent, tongue slightly charged, conjunctiva a little yellow. Epigastrium very tender. No pain on pressing the abdomen. No blood in the vomited masses. Somewhat constipated. Administered a saline laxative, which opened the bowels. Morphia sulph. and sub-nitrate of bismuth relieved the vomiting. Two days later he complained of severe dyspnœa, and had to sit up in bed, became very restless, pulse exceedingly frequent; the extremities became cold, abdomen very tender to the touch. Died in collapse. *Autopsy* forty hours after death. The intestines found much distended with gases; they adhered to each other and to the abdominal walls.

The whole peritoneal surface was opaque, and covered here and there with fibroid membranes, which could be readily detached. In the pelvic cavity about three hundred ccm. of a yellowish liquid, containing considerable fibrous floculi and pus. Within the cavity of the little-dilated stomach a small quantity of reddish-brown liquid and some blood coagula. Externally the organ is of a dark purple color, and all its membranes are thickened and infiltrated, particularly in the pyloric region. The sub-mucous tissue is partly opaque-yellow, partly reddish from hyperæmia, in many places infiltrated with pus, and œdematous, its consistence like jelly. Owing to the great volume of the sub-mucous structure, the mucous membrane is smooth, slightly stretched, very shiny, and covered with yellow, gray, brown, and black spots, which in some localities have clear outlines, in others diffuse. They are due to the action of the acid gastric juice upon the extravasated blood. The muscular and serous coats were less swollen, but very much infiltrated with pus.

The alteration of all the structures indicates that the irritation and subsequent changes originated in the sub-mucous tissue and spread from there upward and downward, and differs from changes taking place in the mucous membrane primarily.

FIG. 2.—*White Softening of the Stomach and Perforation of its Walls.*

CASE.—A babe of eighteen months.

History and Symptoms.—Had measles about three weeks ago and suffered ever since with hoarseness and slight cough. As a number of children in the same house were affected with scarlet fever, it was supposed that this child was also affected thereby, and a physician called in, who took hold of its tongue in order to examine the cavity of the pharynx, when the child suddenly expired. *Autopsy* three days later. The brain and its membranes quite normal. The aryepiglottal ligaments slightly swollen, and in a state of catarrhal inflammation. The same was the case with the larynx, trachea, and bronchi; in their cavities a quantity of tough mucus existed. The left lower lobe of the lung was inflamed. The peritoneum in the region of the stomach was covered with a layer of very sour milk, which escaped from the organ by a fissure through all the coats, of about six centimeters in length, extending along the great curvature. This extensive perforation must have been the cause of the sudden death.

FIGS. 3, 4.—Perforation of the œsophagus and great atrophy of the cardial portion of the stomach, from chronic ulcerations in their walls.

FIG. 5.—Simple circular ulcers of the stomach. They are very shallow, and only involve the mucous membrane. The dark color of the ulcerated part and the injected tissue show that there was very circumscribed local disturbances of the circulation in each affected part. The filled vessels are visible near the ulcers only.

C G, great curvature. *H*, hæmorrhage. *P R*, *P P*, perforations. *O E*, œsophagus. *E*, erosion of the mucous membrane. *H U*, hæmorrhagic ulceration. *U N*, nervous vagus. *D*, duodenum. *M P M*, softened mucous membrane. *P*, pylorus.

Functional Disorders of the Stomach.

These may be divided as follows:

1. *Changes of Alimentation.*
2. *Modification of Phenomena of Digestion.*
3. *Modification in the Processes of Secretion and Excretion.*
4. *Modification of Nerve Phenomena in the Gastric Functions, and its State of Sensibility.*

CLASS 1.—*Alimentary Changes—Disturbances in the Appetite.*—Sensation of hunger may be enormously increased (*boulimia*), decreased (*anorexia*), perverted (*pica*), or totally destroyed.

The inordinate desire for food was formerly ascribed to enormous dilatation of the stomach, and lack of power in the pylorus to close, *pyloric incontinence;* termination of the ductus chaledochus into the stomach; hypersecretion of the gastric juice; an unusually short intestine, worms, etc. Vesalius has, for instance, found the bile duct ending in the stomach of a certain glutton. Another celebrated great eater was found by Percy to have an enormous sized stomach. Cabral found in a certain polyphagist, his former patient, an enormous stomach, followed by an exceedingly short intestine. To the anatomical peculiarities of those individuals were of course ascribed, in those non-critical days, their ravenous appetites. Still, there is one interesting fact connected with this matter, that all exceedingly voracious carnivorous fishes have enormously-developed stomachs and but very short intestines. Ravenous appetite in man is undoubtedly due more to a state of incomplete nutrition, assimilation, and nervous disturbance than to any peculiarity of the stomach.

Gastric irritation may sometimes produce boulimia, especially in a chronic state, and often furnishes a valuable symptom, when the great desire for food is soon followed by anorexia, or distaste for it. Very many dyspeptics have a peculiar gnawing sensation in the epigastrium, which they mistake for intense desire for food. Growing children, convalescents from exhaustive diseases, and pregnant women furnish examples of boulimia with which gastric irritation has nothing to do.

Anorexia is the most common phenomenon associated with all sorts of gastric derangements. There are a number of very grave diseases of the stomach, such as softening, ulceration, and induration of the sub-mucous tissue, in which there are no other symptoms than total loss of appetite. Anorexia sometimes exists in very insignificant diseases of the organ, as post-mortem examinations, made by very competent persons, have proved that the causes of this loss of appetite may be due to disturbances outside of the stomach. In fact, it is always found as a constant factor in severe acute and chronic inflammation of nearly every important organ of the body, more especially in such derangements which give rise to disturbance of circulation in the organs of the body. There are depressed conditions of the body when every function is almost at a standstill, and when the processes of life are at so low an ebb that no metamorphosis of the tissues takes place, nor does even their nutrition go on; in such a condition, complete anorexia exists. This constitutes the state of *Inanition.*

Pica, perverse appetite, is very common in many diseases, and is only indirectly connected with the stomach.

Pregnant women, chlorotic young persons, hysteric individuals, and many persons affected with mental diseases often manifest the strangest desires for food or drink. The dirt-eaters in Africa and South America exemplify such ethnic perversity of appetite. In great losses of blood, during the shock from mechanical injuries, in fevers, and fatigues with exhaustion there is always an intense desire for drink, not to speak of the craving for pungent and highly stimulating substances, which in the very fewest of cases only can be attributed to direct gastric lesions.

CLASS 2.—*Alteration in the Process of Digestion—Dyspepsia.*—It is unnecessary to state that dyspepsia is one of the chief symptoms of actual gastric lesions, and even irritations. Yet, like the above-described derangements, dyspepsia may be due to disturbances lying outside of the stomach. Clinical histories have often enough revealed the apparently singular fact that in very many cases of severe dyspepsia of long standing no important anatomical changes in the organ have been found after death, while very deep alterations in the tissues have produced very insignificant disturbances of digestion during life. Even in those cases where distinct lesions of the organ were found, it could not always be proved that the lesion and dyspepsia during life stood to each other in direct relation of cause to effect.

For clinical purposes alterations of the processes of stomach digestion are classified according to the discernible causes producing them. These are: 1. Such as have actual acute or chronic alterations of the parenchyma of the organ. 2. Those in which there are derangements in the secretions of the organ from irritation, such as hyperæmia, etc. 3. Those caused by asthenia, either from insufficient circulation, insufficient innervation, or mechanical obstacles. 4. Strictly nervous derangement, either of the organ itself, or by reflex from other organs or from the centra. Nervous dyspepsia, the most common form of dyspepsia, is that which is either caused by direct irritation of the several tissues of the stomach, or by partial or total atony of the organ. Such atony is liable to be produced by any exhaustive process going on in the body. In asthenic dyspepsia there is usually no thirst; the tongue is pale and very lax. After taking food the patient experiences a sensation of fullness and weight in the gastric region, often a feeling of great tension. The pulse is usually not irritable, but is sometimes apparently full but very compressible, from the great loss of elasticity in the arterial coats and low degree of blood pressure. Vomiting seldom exists, and then only when the organ is overloaded or distended by liquids or gases.

Skorczeski (*Ætiologie und Pathogonse, Denkschrift, Warschau*) divides the causes of asthenic dyspepsia into three classes: 1. Such as produce direct irritation upon the mucous membrane. 2. Dyscrasis and anomalies in the blood. 3. Central nervous disturbances. Of the latter varieties of disturbance of digestion, he observed sixty-four clinical cases, thirty-four of which owed their origin to psychical derangements, twelve to uterine affections, three to onanism, and three to chlorosis. To prove the connection of those disturbances with gastric atony, he bases his explanation upon physiological grounds, claiming that they act upon the splanchnic nerves in such a manner that they produce contraction of the

TABLE VI.

GASTRIC LESIONS.

FIG. 1.— Partial stenosis of the pyloric opening from enormous hypertrophy of the muscular coat of the stomach in the pyloric region.

P, pylorus. *D*, duodenum. *T M*, muscular tissue. *T F*, fibrous tissue.

FIG. 2.—Partial stenosis with incontinence or insufficient closure of the pyloric orifice, from enormously hypertrophied mucous membrane in the pyloric region, following very deep and very extensive ulceration of the tissues. The hypertrophy is here not only of the highly-thickened and condensed mucous membrane, but also an enormous enlargement of portions of the sub-mucous structures, raising the mucous membrane into high and extensive folds, which project into the cavity of the organ. It constitutes on one hand an obstacle to the free passage of the digested food into the intestine, thereby producing dilatation in the cardial region; on the other hand, insufficient closure of the opening, thereby permitting the bile and pancreatic juice to penetrate from the duodenum backward into the stomach, interfering with gastric digestion to a great extent.

In the œsophageal portion and the cardia there is strong constriction in the walls, a remnant of extensive cicatrization in that region. The cicatricial tissue is very dense, partly of cartilaginous consistence, utterly devoid of any glands or follicles, and hence useless for digestive function.

E, ectatic state of the cardia. *P*, pylorus. *E*, erosion. *C*, cicatrix.

FIG. 3.—*Pigmentation and Cicatrization of the Mucous Coat of the Stomach.*

The membrane is partly restored from extensive ulceration, but has lost many of its glands and follicles, especially in the fundus and the pyloric region. It has a striped appearance and is still partly indurated, so that, with the loss of a quantity of its functional tissue and the dense condition of some of the remaining, the work of digestion can be carried on very imperfectly. The obliterated portions were never fully re-established, and a very thin single layer of epithelium took the place of the normal functional tissue.

C, cicatrix. *D*, duodenum.

FIG. 4.— *Extensive Dilatation of the Stomach, with Massive Non-malignant Hyperplasia of the Mucous Membrane.*

When hypertrophy of the glandular layer of the membrane reaches a degree that it finds no more space upon its sub-mucous basis, it develops into ridges and depressions, following the wavy lines of the krypts and follicles.

In this case the enormously-enlarged membrane in the pyloric region has still retained its physiological character, but in the fundus, which is exceedingly dilated, it formed into a number of polypi, which are useless for functional purposes. By its folds assuming an areolar arrangement, there are found, a little distant from the pylorus, a number of places where the glandular layer has reached the highest degree of hypertrophy. A number of spheroid projections are formed above the surface, which, by still further increase, assume a longitudinal shape, and from this, by still further growth, pass into pedunculated or non-pedunculated polypi. The head or free extremity of those vegetations usually increases in breadth and forms club-shaped excrescences, several of which have a common base. They disfigure the membrane, and, when there are many of them, disturb digestion. Internally the polypi are made up of enormously-dilated gastric glands, and occasionally of real cysts, containing yellowish serum or mucus. Their interglandular tissue constitutes peculiarly cloisoned network of dense connective-tissue, occupying a considerable space of the membrane.

O E, œsophagus. *V P*, polypous excrescences. *M M*, mucous membrane. *F*, follicles.

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intestinal vessels and atrophy of the walls of the stomach. The result of it is incomplete and retarded absorption of the partly-digested food, and derangement of the secretory and excretory functions. Dilatation of the organ, habitual constipation, lack of assimilation, feeble trophic state of tissues, and a variety of nervous disturbances will follow.

Digestive troubles from chlorosis are principally characterized by neuro-muscular phenomena, such as epigastric pains, cramps, sometimes vomiting; very seldom dyspepsia from secretory derangements, and still more seldom are there any organic lesions or simple ulcers. These neuro-muscular disturbances are not different from simple gastralgia or nervous vomiting, except that there is a total absence of vascular murmur and good color of the integument in the latter. (*See, Leçons de Patholog. Experimen.*)

CLASS 3.—*Dyspepsia from Disturbance of Gastric Secretion.*—It is manifested by a variety of local and general symptoms, and is very liable to assume a very chronic form. The proximate cause is always alteration of gastric secretion from the mucous membrane. *Leube* (in *Clin. Lecture, No.* 62, *Volkmann's Collection*) designates it as *gastric torpor*, due in the great majority of cases to insufficiency of secretion of muriatic acid, very seldom to an inadequate quantity of pepsin. There is very often an insufficient supply of gastric juice *in toto*, or a hypersecretion of mucus, and little or no effective gastric juice. Occasionally there may be a very frequent pulse, but it does not indicate any gastric irritation.

In the above-described diseases, where some of the deepest changes are found in the stomach after death, there are sometimes during life the most insignificant functional disturbances; especially vomiting, a symptom that might prove of the highest diagnostic value, is very frequently absent. Even when vomiting exists, it can not always be ascribed to the lesion located in the stomach itself, for it is very often due to causes existing outside of the organ.

Besides the food and drink, the substances vomited may be mucus, blood, bile, muco-bile, or sometimes even pus. Local causes of the vomiting are the substances, in quantity or quality, existing in the organ, and keeping up the irritation produced by them. Some people vomit regularly every morning. The substances ejected are usually glairy mucus and some bile, or a highly watery yellowish liquid of neutral or slightly alkaline reaction, containing some biliflavin or biliviridin. The appetite of such persons for their morning meal is hardly ever diminished. Rene Prus considers it due to accumulation of large quantities of mucus secreted during night in the stomach from hypertrophy of the mucous membrane in the cardial region. He calls it catarrhal *gastrorrhœa*.

The increase of secretion of mucus in the stomach when empty must undoubtedly be due more to a state of constant irritability, which acts more or less steadily upon the secretory apparatus, than to mere hypertrophy of the secretory glands; as in the salivary glands, hypersecretion can only be due to derangement of the trophic nerves (either irritation of the vaso-dilator, or paralysis, permanent or temporary, of the vaso-contractors). Pregnancy, sea-sickness, many narcotic poisons, and emetics act by reflex irritation upon the secretory apparatus through the trophic nerves. The newly-forming epithelium of the mucous membrane is very soon lignified by any high degree of hyperæmia existing in the tissue, and soon will undergo desquamation, fatty and granular changes, and become a component of the great quantity of liquid oozing through the porous layer of the external stratum of the basement membrane. Such blennorrhœa—for such it really is—is always the first symptom of alcoholism, gastromalacia, and many forms of slowly-developing changes of the mucous membrane, which, if not promptly checked, will sooner or later degenerate into a malignant lesion. Irritant poisons produce vomiting, which continues for some time even after their passage from the organ, unless the tissues of the stomach have at once been destroyed by their contact. Acute peritonitis always induces vomiting. In its chronic form, especially when the serous layer of the stomach is affected with extensive hyperplasia, vomiting always occurs. In acute peritonitis, with extensive vomiting, the inner coats of the stomach are very often found, after death, in an almost normal condition, possibly slightly injected.

For outside causes of vomiting we must look to the nervous system, chiefly the nerve centers. *Magendie* has long ago demonstrated this fact by removing stomachs of small animals and skillfully substituting in their place hogs' bladders. By introducing tartrate of antimony into those animals' veins, efforts at vomiting, and sometimes even moving of the inside of the bladder took place. In fact, vomiting is one of the instinctive nerve-actions to protect the body against the injurious substances introduced into the alimentary organs or into the blood, for not only does the stomach eject the offensive substance placed in its cavity, but it also carries on for a time the function of excretion, and eliminates quite a good portion of many injurious substances introduced into the blood. Arsenic and similar poisons, when already absorbed in the blood or when directly placed into it, produce excretion of the substances from the blood by the whole intestinal mucous membrane, besides the mechanical act of emptying its contents. In very many diseases of other organs (nephritis, lepto-meningitis, sub-arachnoidal meningitis, etc.) vomiting is a constant phenomenon, without, however, the existence of any gastric lesion. Sudden mental disturbance and hysteria are very often accompanied by vomiting.

CLASS 4.—*Modification of Sensibility.*—The gastric mucous membrane is but little sensitive to touch, as Richet has proved by introducing food through the fistulous opening of his patient, Marcelin, affected with gastric fistula (*Journal de l'Anatomie*, 1878, p. 170; also *Beaumont, Experiments on the Canadian Hunter*). On the other hand, many substances and sudden change of temperature produce distinct and often painful sensations upon it. As its function is essentially based upon reflex activity to and from several nervous centra, it is very prone to be irritated and affected by very varied reflex effects; likewise is it possible to produce many reflex phenomena from the coats of the stomach upon the viscera, the circulation, respiration, and the cerebro-spinal and sympathetic centers. Meyer and Pribam's experiments have revealed the fact that a great rise of arterial pressure and slowness of pulse are produced by irritating the walls of the stomach. They describe it as follows (in *Studien zur Pathologie, Session of the Imperial Academy of Science, July*, 1871): "When the vago-sympathetic nerve-filaments of the neck were cut through, there was no slowness of pulse; but the increased arterial pressure remained. Irritation of the stomach walls was produced in a variety of ways, by exposing the opened organ to the air, by the introduction of a sound through the mouth, and by electricity. When a current sufficiently

STENOSIS OF THE PYLORIC ORIFICE.
EXTENSIVE DILATATION OF THE STOMACH.

Sec. VI. *Tab. VI.*

Fig. 1.

Fig. 2.

Fig. 3.

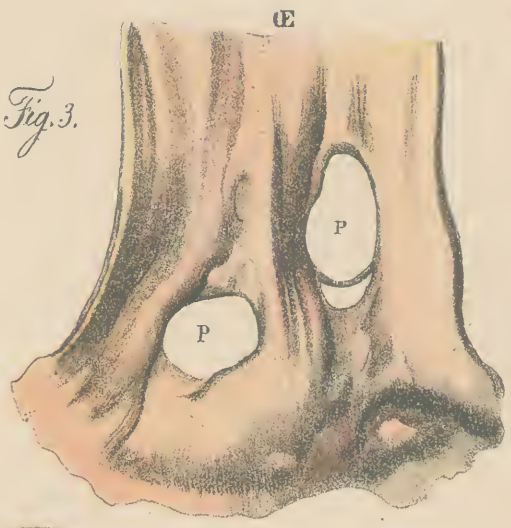


Fig. 4.

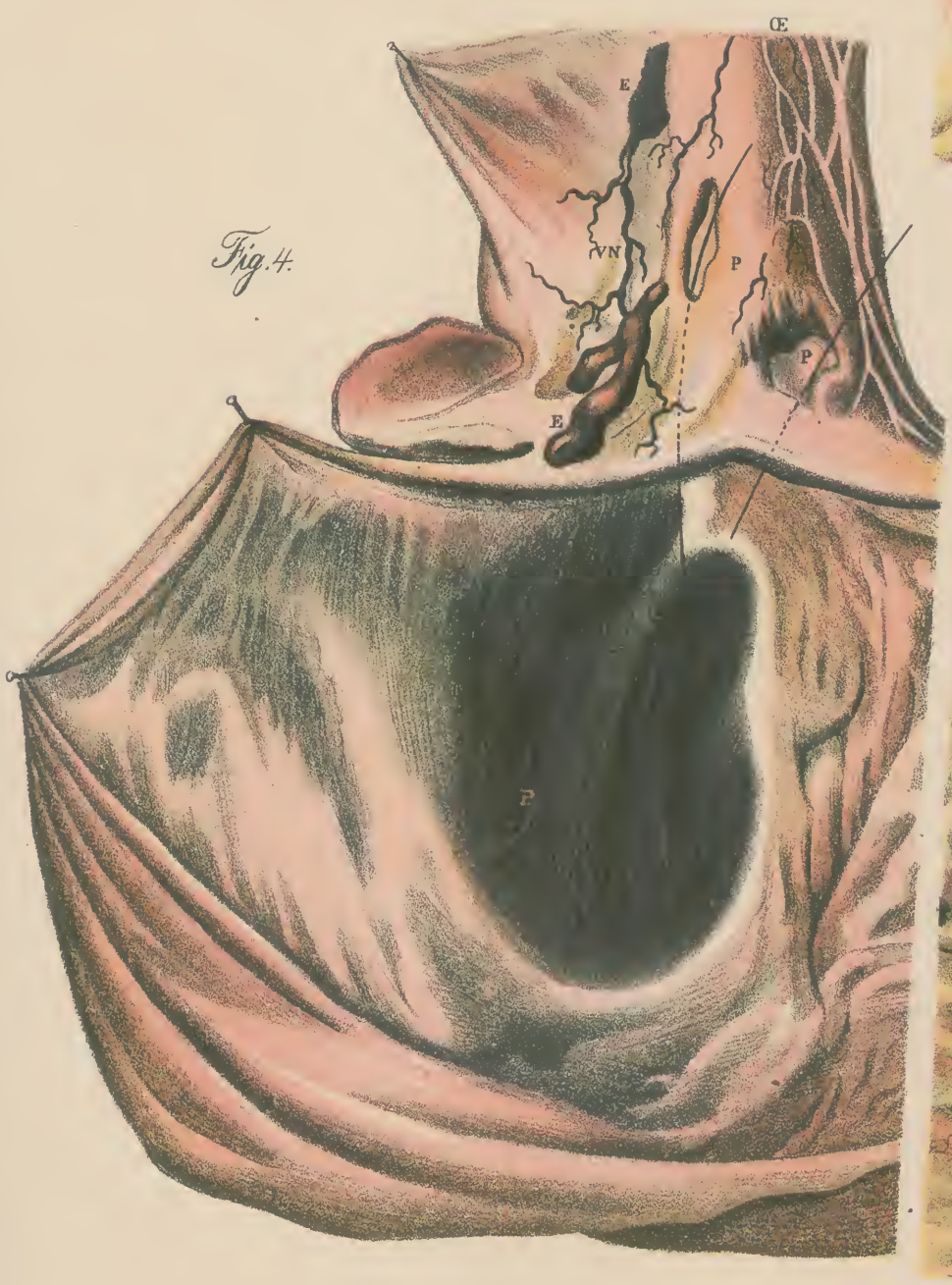
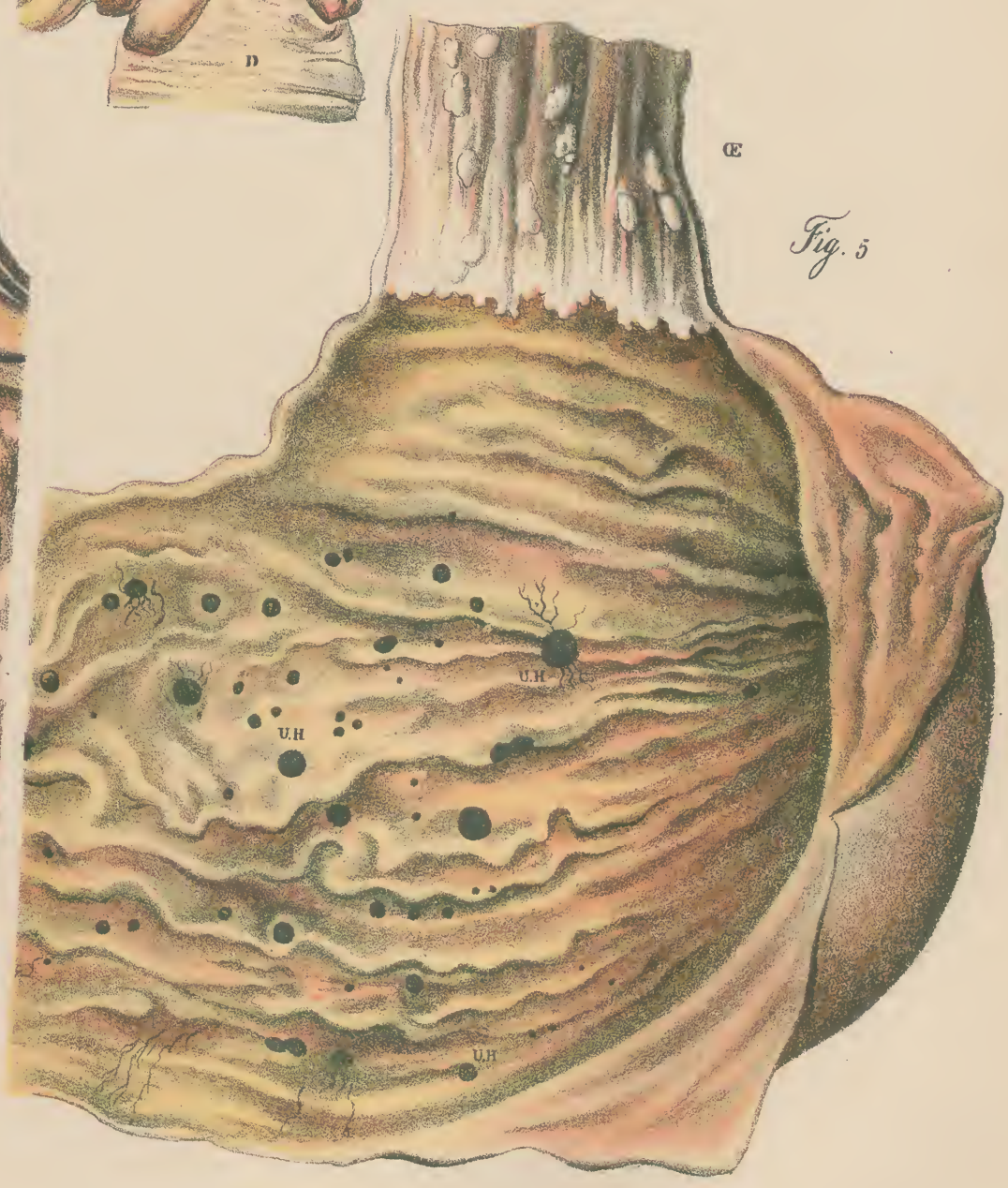


Fig. 5



Sec. VI. *Tab. VII.* **SOFT CARCINOMA. CARCINOMATOUS ULCERS. ADENOMA OF THE STOMACH.**

Fig. 1.

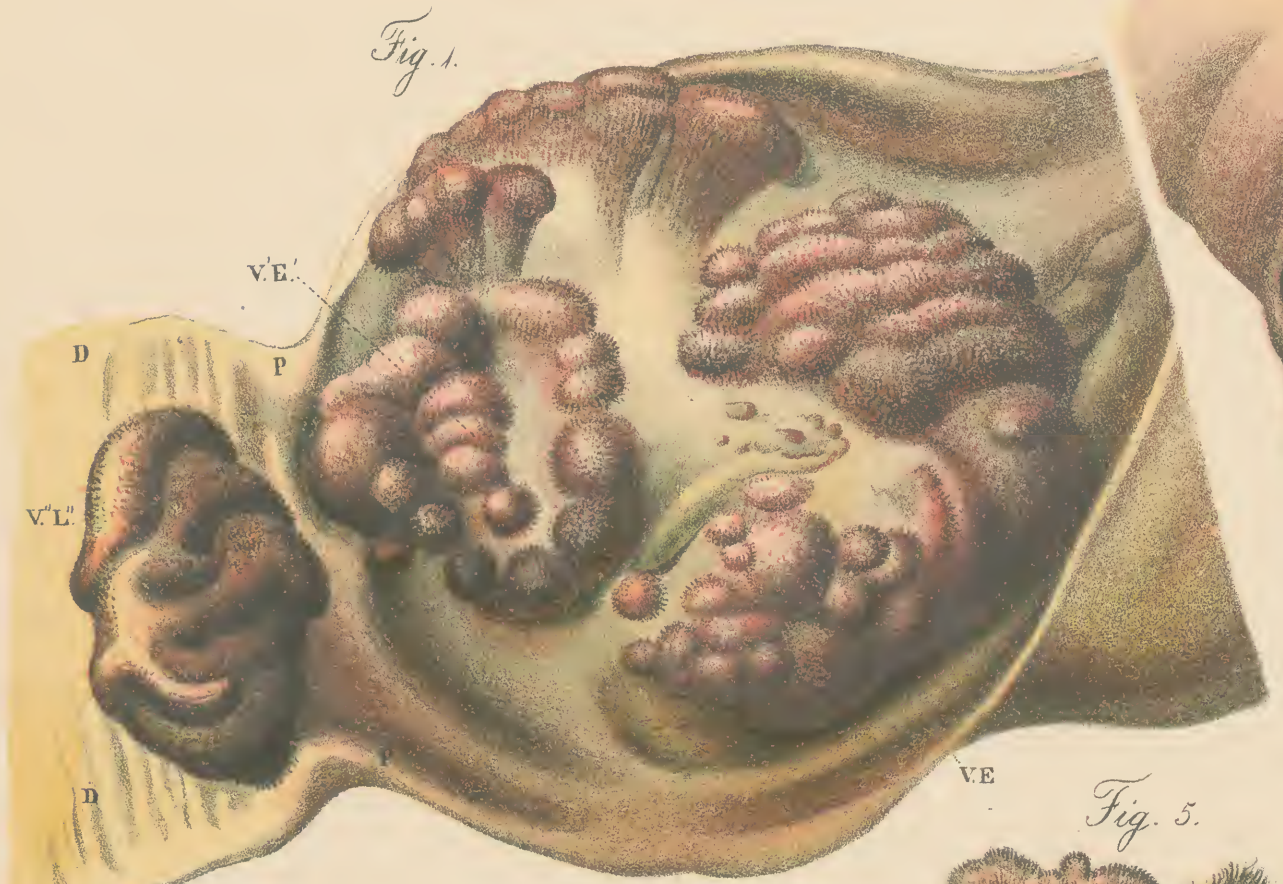


Fig. 4.

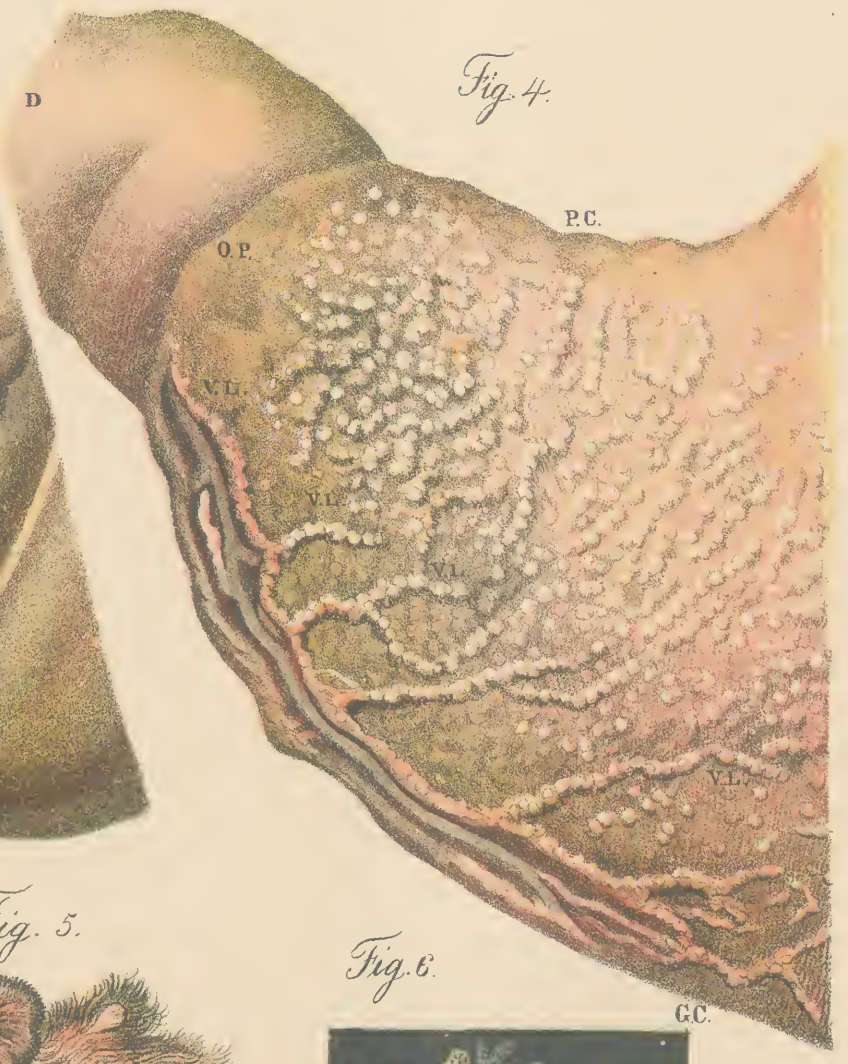


Fig. 5.

Fig. 6.

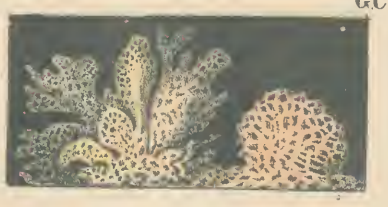


Fig. 2.

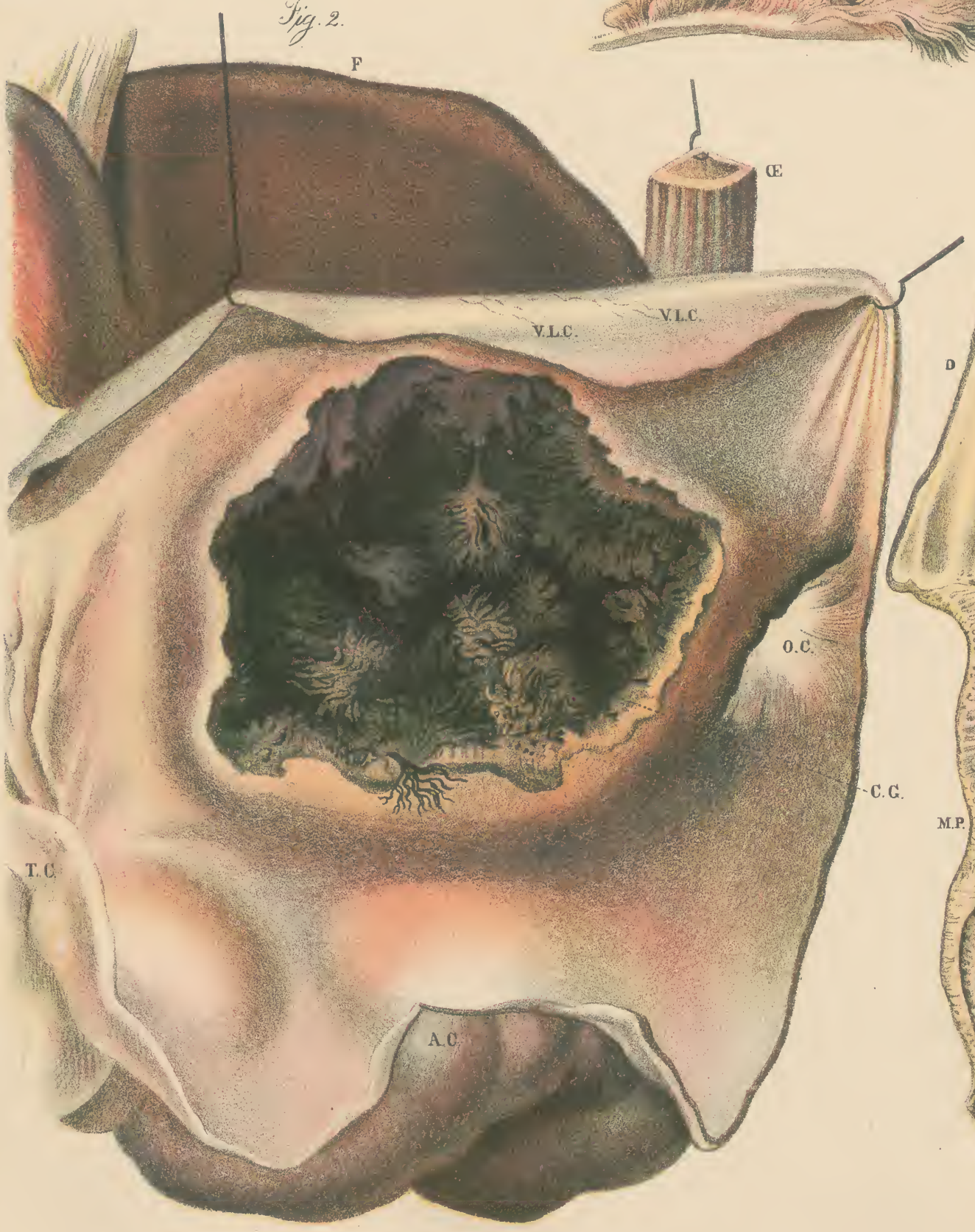


Fig 3

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TABLE VII.

FIG. 1.—*Soft Epithelioma* (*Medullary Carcinoma*) *of the Stomach.*

Fig. 1 shows this cancer, several inches in extent, and involving the pylorus and portion of the duodenum. The disease commenced in the lesser curvature and descended partly upon the anterior, partly upon the posterior aspect of the organ. Taking its origin in the epithelial covering of both the glandular and interglandular structures, it formed a number of fungiform tumors, some soft, some hard, and infiltrated not only the mucous, but also the sub-mucous tissue, causing, by the great pressure it exerted upon the tissues, destruction of both. The histology of this cancer is described by *Thierfelder* as follows: "The fibrous strings of the *stroma* contain great numbers of spheroid and spindle-shaped connective-tissue cells, and form tubular sacs in which are contained the polygonal cancer cells, composed of a very granular and fatty protoplasm, and supplied with a large, very distinct nucleus. They remind one of the cells of the peptic glands, yet it can not be proved that the latter originate the former." There are a number of small blood vessels traversing the neoplasmata. After it has reached a certain size it undergoes ulcerative destruction, leaving behind a number of fringes which still adhere to the fundus of the ulcer. They are remnants of the *stroma* of the cancer, which resists destruction far longer than the cancer cells. By the action of the gastric juice they become gradually detached in pieces from their bases, and produce, by exposing the vascular layer, those hæmorrhages characteristic of soft cancer; that is, the blood will be vomited in soft clots, having a more or less honeycombed arrangement and of the color of coffee-grounds or dark chocolate.

A section of the papilliform growth in Fig. 5 of this Table shows the raised mucous membrane, the enormously hypertrophied structure and fibrous arrangement of the stroma, studded on top with epithelial spikes visible to the naked eye. Fig. 6 of this Table shows a section of the wall surrounding a carcinomatous ulcer from which the cancer cells have been removed by destruction, leaving only the spongy stroma, which has its basis on the basement membrane, while only debris of the atrophied glandular layer represent degenerated and deformed mucous tissue.

FIG. 2 represents the fundus and the walls of a carcinomatous ulcer in the region of the coecum. The surface of the fundus is black (the color is due to the action of the enteric juice upon the tissue), the walls are jagged, and a number of fringes are projected and float upon the surface of the ulcer. The deeper layers of the edges of the wall are darker than the more external.

FIGS. 3, 4.—*Adenoma Distruens.*

This form of cancer, although developed in the mucous membrane, does not produce its hyperplastic action upon the outer surface of the membrane, as in the inner strata, upon the sub-mucous tissue, the muscular and the serous tunics. Within these, and even upon the outer surface of the fibrous coat, it develops into innumerable solitary nodules—very rarely aggregated—which follow the course of the internal and external lymphatics. To the free surface of the mucous membrane within the cavity it imparts a pitted and honeycombed appearance, with here and there a few raised vegetations. All the walls of the organ are thoroughly infiltrated; the mucous membrane especially is thickened and much increased in volume. The pyloric region is peculiarly enlarged. The great inclination of this form to produce metastatic deposits is here shown by the broadcast carcinomatous nodules upon and within all the structures. Along the great curvature and within the pyloric region the veins are filled with cancerous thrombi.

P C, small curvature. *G C*, great curvature. *O P*, orifice of the pylorus. *V L*, lymphatic vessels. *O E*, œsophagus, *D*, duodenum. *V E*, raised vegetations. *F*, liver. *T C*, transverse colon. *C G*, fundus of ulcer. *V L C*, coecal valve. *A C*, ascending portion. *M P*, pyloric portion. *M M*, mucous membrane. *C M M*, carcinoma of that membrane. *V*, vegetations..

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strong to produce contraction of the muscular wall was applied both high arterial pressure and a slow pulse were produced. The splanchnic nerves were not at all affected. Neither the influence of these vaso-contractors nor of the contracted vessels of the contracted muscles could here be the cause of the arterial pressure. By pinching the gastric walls with a forceps the same phenomena of circulation was produced." "In order to produce a reflex effect from irritation of the walls of the stomach similar to that which might be produced by disturbance of digestion, the stomach was dilated to an excessive degree by means of a soft rubber ball which was dilated in the organ by introduction of air into it through a catheter in any desirable quantity. Distention of the stomach by this means yielded, in a clearly-defined manner, a great rise of arterial pressure and a slow pulse. These experiments prove that the reflex upon the heart and circulation from the stomach are brought about by the nervous centers. These reflex actions resemble those which are produced by irritations of sensory nerves, existing either on sensitive surfaces or in the muscular tissue . . . . From these experiments we are justified in concluding that the nerve-filaments from which reflex action upon the heart and circulation can be exerted are situated in the muscular and serous coats of the stomach." A practical lesson from these experiments can be drawn in this way: That distention of the stomach produced by partly-digested food, overfilling of the stomach with too much and too hastily taken food or with indigestible material, is capable of producing so slow a pulse as to interfere with the steady aeration of the blood, and producing so high a pressure as to cause hyperæmia and hypersecretion in the stomach. On the other hand, the contraction of the small blood vessels during the high arterial pressure may produce such anæmia of the brain as to greatly impair its functions, especially the innervation of the intestinal canal. Headache, vertigo, a feeling of coldness in the upper and lower extremities, and many more such symptoms of disturbance of the nerve centers, are very common in functional disturbances, especially in distention of the stomach.

Regarding the slight sensibility of the gastric mucous membrane very many examples can be produced where the membrane was highly injected, softened or indurated, or even ulcerated, without any feeling of pain beyond a slight colic or insignificant sensation of malease. The abdominal wall may be roughly passed over or rubbed without producing any pain in the severely-affected intestine. Only when the ulceration has penetrated into the muscular and serous coats and is on the point of perforation, is intense pain produced. Pain from nervous irritation may sometimes exist in the stomach without the least alteration of the tissues of the walls (gastralgia). For instance, people who are, by their calling or otherwise, subject to very sudden changes of temperature often suffer intense pain in the stomach without any perceptible cause existing in the walls of the organ. Gastralgic attacks, described by Delamarre and Chareot as *crises gastric*, are very common in dorsal tabes. Such patients are often attacked with intense gastralgia, associated with vomiting, strangling, nausea, dizziness, and sometimes with diarrhœa, but usually with very obstinate constipation; sometimes even swelling of the abdomen; rigors of the severest kinds, convulsion, trembling, vomiting of mucus, blood, or bile; very frequent pulse. Such attacks may last a day or two, may return weekly or even daily, and gradually reduce the flesh and the health of the affected person.

See describes a form of *hyperæsthesia* and painful contraction of the abdominal muscles, which simulate certain phenomena found in ileus or peritonitis, and is often associated with actual disturbance of the stomach digestion, which he called pseudo-gastralgia. These are all produced by general anæmia. This hyperæsthesia seldom assumes a neuralgic form. Ordinarily there are muscular pains situated in the upper portion of the rectus abdominalis muscle in the epigastric region, often resulting in a permanent contraction of the muscle, and when the person is in a vertical attitude, or any tension upon the muscle be produced by the distended wall of the stomach, the pain becomes excruciating. The pain in that region, and especially when there is actual disturbance of digestion, will often mislead the physician in his diagnosis. Lessening of the pain when the tension is removed by the person's lying down or relaxing of the muscle will readily indicate the differential points. Pressure upon such painful muscles will *always* increase the pain, while when the pain in the epigastrium proceeds from a diseased stomach pressure will increase the pain *only during digestion*. This muscular pain may, however, be the consequence of a disease of the stomach, the same as diseases of the thoracic organs produce pain of the thoracic muscles. Of course, the origin of the reflex action upon the muscles must necessarily be traced to its proper source.

Derangement of Gastric Innervation.

Although not as frequent as other nervous disturbances of digestion, there is a form of nervous dyspepsia characterized by a number of symptoms, chief among which is very severe pain in the stomach after meals. The pain is sometimes so intense that the affected persons abstain from food for fear of the intense suffering that follows eating. At the same time there is a desire for food, for the appetite is in such cases rather heightened than lessened. Both starvation and intense suffering are the alternate miseries of such afflicted people, and both or either reduce them to a low anæmic state. The pains either follow the meal immediately or take place an hour or two later. In the latter case the persons are seized with a sense of extremely painful constriction or spasm in the gastric region, associated with a keen desire for food or a sense of emptiness in the stomach. Very rarely is the food vomited immediately after eating.

When the disease assumes this form the person is threatened with a dangerous dyspeptic *inanition*. In malposition of the fœtus and in exceedingly sensitive and highly irritable uterus of pregnant women, this form of dyspepsia usually causes very dangerous disturbance of nutrition. Anæmic persons are usually affected with this form of disease, for the insufficient quantity of nutriment in their blood readily favors *denutrition* of the nervous system, and gives rise to hyperæsthesia or anæsthesia of the different organs. In all cases of this kind there is hardly ever any important change in the gastric secretion. The irritation seems to be exerted by the food upon the otherwise not very sensitive mucous membrane of the stomach, which now becomes exceedingly hyperæsthetic. The irritation is carried by the splanchnic filaments to the spinal marrow and brain, and from there reflected by the motor fibers upon the muscular coat of the stomach by the pneumogastric and phrenic, from the stomach and diaphragm upon the muscles of the abdomen. Cramps, spasms, and vomiting are thus produced. As innervation has little to do directly with the quality of the secretion of gastric juice, there is therefore no change in the liquid, and what food stays in the organ usually undergoes although slow yet perfect digestion. (*See, Leçons de Patholog. Exper.*)

TABLE VIII.

FIGS. 1, 2.—*Gelatiniform Cancer* (*Colloid*).

Fig. 1 represents an enormously infiltrated and exceedingly hypertrophic condition of the whole organ, cancerous masses forming nearly parallel ridges, made up of innumerable papilliform excrescences crowded by the side of one another. They are nearly transparent and have a jelly-like look. The carcinomatous process has here passed upon the lesser and greater omentum, and scattered upon them are very many nodular tumors having a fine vascular layer outside and consisting chiefly of fatty detritus and cancerous infiltrations. The stomach wall, chiefly in the œsophageal region, is indurated, and has an extraordinary thickness. Outside it is embossed with many cancerous tumors.

Fig. 2 represents a portion of the integument covering the umbilicus. Through the opaque white peritoneum there is a hernial protrusion of the omentum (*H O*). The sac has an embossed exterior like the outer surface of the stomach. Above the umbilicus another sac of nearly similar appearance is observed (*H L D*). The sac (*C*) is more of the form of a cyst. The serous membrnae (*S*) in the umbilical ring is also covered with tumors.

FIG. 3.—Excessively contracted and deformed stomach from cancerous ulceration and atrophy of the tissues. The spleen (*R*) is attached laterally to the cardiac portion, and has drawn the organ in that direction out of its position. The œsophageal opening is very narrow (*O C*). The greater and lesser omenta (*E P*, *E G*) are thickened and covered with cancerous tumors.

FIGS. 4, 5.—*Fibrous Carcinoma* (*Scirrhus*).

Fig. 4 presents the pyloric region in a state of diffuse induration and increase of volume of all the tunics of the organ. The internal surface is covered with hypertrophic tissue and partly sub-mucous tough fibrous bands or lamina. All the coats are still recognizable, but show enormous increase of volume from abundant infiltration.

Fig. 5, a section showing the cancerous masses imbedded in the sub-mucous tissue.

Motor Neuroses of the Stomach.

When the muscular tissue has lost its contractility, or when the stomach becomes immovably attached to the abdominal walls, there is diminution of peristaltic action of the organ. On the contrary, the peristaltic action is sometimes exceedingly increased, and becomes visible and palpable through the abdominal walls. *Kussmaul* describes it as "*peristaltic restlessness of the stomach.*" This occurs in cases of very dilated stomach, with strong hypertrophy of its muscular coat, in stricture of the pylorus or the duodenum by closure of the pyloric orifice with malignant or non-malignant growths. This hypertrophy of the muscular tissue is compensatory (similar to compensating hypertrophy of either ventricle of the heart). It is gradually produced, in measure as the food meets with obstacles in its passage through the pyloric opening.

In purely spasmodic contraction of the pylorus, even the normal muscular tissue will sometimes produce exaggerated peristaltic action, to overcome the pyloric resistance, but of course it will not be as well pronounced as in muscular hypertrophy. Irritation of the stomach filaments of the pneumogastric nerve, or even portions of the solar plexus which connect with the muscles of the stomach, further hyperæsthesia of the mucous membrane, mechanical irritation, certain poisons, direct or reflex irritation within the nerve centers, etc., all produce this affection. *Kussmaul*, in describing this disorder (in the 181st *Clinic Lecture*, *Volkmann's Collection*), cites a number of cases of the kind, and especially mentions one of a woman who became affected with almost incessant peristaltic action of the stomach to an insupportable degree, and giving her no rest even in her sleep. Only when her stomach was perfectly empty did she have a few minutes' peace. The primary cause of her disorder was poverty and domestic trouble. She recovered after two years' hygienic treatment. The same author thinks that peristaltic derangement may exist even when there is no pyloric stricture, but simply *hyperkinesis* of the muscular tissue brought about by long use of drastic purgatives and other substances capable of irritating the peristaltic centers. *Busch* (in *Virch. Arch.*, *Vol. XIV*, 1858, p. 150) describes a case of that kind where the peristaltic movement would last all day, but would stop in the night. *Magendie*, *Longet*, *Schiff*, who have experimented upon dogs' stomachs, found that as long as any chyme was contained in the cavity the peristaltic action took place, but was perfectly quiet when it became empty. It has been found that many tumors and other neoplasmata are capable of producing similar disturbances. As many cases of hyperplasia of the adjacent organs are capable of producing stenosis of the pyloric opening or stricture in the duodenum and thereby dilatation and hypertrophy of the stomach, peristaltic derangements in such cases are very natural.

Gastric Trouble from Causes in the Nerve-Centers Direct and by Reflex Action.

In his repeated experiments upon the nerve-centers which Schiff has described (in *Arch. f. Phys. Heilkunde*, 1846, p. 677; *Ueber. d. Gefaessnerven*, *Arch. f. Phys. H.*, *Vol. XIII*, p. 30, 1854; *Leçons sur Phys.*, *Vol. II*, 1867, *Chap. XXXV*) a number of gastric disturbances ensuing from severing the optic thatamus or the cerebral peduncle on one side. The gastric lesions produced by the injury of those portions of the brain consisted in hæmorrhagic infiltration and softening of the coats of the stomach. The severance of the central conducting fibers of the vasomotor centra produced neuro-paralytic hyperæmia of the mucous coat of the organ. Cutting through the pons Varoli on one side, as well as one half of the medulla oblongata (especially downward toward the lower point of the *calamus scriptorius*), the same effect upon the stomach was produced. Farther down in the spinal marrow he could not produce any stomach trouble by a section of the conducting fibers. He concluded that the vasomotor fibers of that viscus must enter the spinal marrow above the dorsal portion. He was farther led to the conclusion that *all* peripheral branches of the sympathetic contained vasomotor filaments of the stomach, for when he destroyed a few of these only and not all, there was no disturbance, or at least but very little vascular derangement in the organ. On the other hand, when he irritated any of these splanchnic fibers, and especially those from the coeliac ganglion, contraction of the vessels of the mucous and serous membranes was produced. *W. Ebstein* states that when he injured the anterior tubercula quadri-gemina, even in a very circumscribed manner, he found the following changes in the animal's stomach immediately after killing it, seven days after the operation: "The mucous membrane was covered with mucus, of grayish-white color in the pyloric region, brownish in other portions. In the fundus there was a shallow erosion; the sharp edges and fundus were pale. The ulcer was irregular in shape. Quite a number of ecchymoses of small size were found in the rest of the membrane." Ebstein further states that quite a number of experiments have proved that one-sided injuries of the cervical medulla produced far greater changes in the circulation of the stomach than those of portions of the brain. But where he severed the *whole* spinal marrow in the cervical region, those changes were either very insignificant or did not take place at all. This author and *Gruetzner* have also produced hæmorrhages and hæmorrhagic infiltration into the coats of the stomachs of animals by irritating the lingual or sciatic nerve several minutes in succession with intervals of a few minutes. When they injured the labyrinth or destroyed the semi-circular canals of small animals (without injuring the cerebellum, according to the method of *Gattstein*), the same gastric lesions were produced. It seems that almost any severe, painful injury of any sensitive surface or of a sensory nerve stump produces the same gastric phenomena. When all the circumstances are examined under which those changes of the tissues of the stomach are brought about, it will be found that the vessels, both arteries and veins, of the walls of the organ, are enormously dilated and turgid with blood, and the volume of the structures enormously increased. Very few lymph corpuscles, but enormous numbers of blood corpuscles are extravasated. The pyloric region is thus mostly affected. Upon such congested tissue the gastric juice very soon produces its corrosive action. If such a condition continues for some time, perforation of the organ takes place, and death follows. All those phenomena can be reduced to the single cause, *exceedingly* high *arterial pressure*, *which produces the most deleterious effect upon an organ normally prone to hyperæmia*. For in all those nervous derangements experimentally produced there were exceeding irritations effected upon the vasomotor centra, and disturbance of the respiratory centra, circumstances which always lead to enormous arterial pressure. When the whole medulla spinalis is severed in the cervical region, there is such sudden and quick fall of the blood pressure that no hyperæmia is produced, and may be compared to that state where very profuse hæmorrhages have greatly reduced the volume of blood in the body. Comparing the above experimental data with many *necroscopic* findings in the stomachs of persons affected during life with mental and nervous diseases, the very legitimate inference might be drawn that connection of the gastric lesions with disturbances of the trophic or vasomotor centra were those of cause and effect.

Further clinical observations are made daily that persons suffering from many forms of neuroses, especially of such organs to which are distributed nerves from the medulla oblongata, cervical and upper thoracic portions of the spinal marrow, suffer nearly always from severe gastric and gastro-enteric disturbances. The older pathologists, such as Andral, Bright, and many more who were at the same time pathological anatomists, have readily connected gastric lesions with mental and nervous diseases, through the instrumentality of disturbances of circulation.

Displacement and Change of Form of the Stomach.

When this organ is perfectly empty it is uniformly contracted and loses much of its curved form, and its caliber is then not much greater than that of the colon. The mucous membrane is then folded in a nearly longitudinal direction. During long abstinence from food or after copious vomiting it assumes the same form. In many diseases it takes a perfectly horizontal position. When mechanically much injured, or when affected with cancer in its middle portion it usually increases in size. Destruction of the fundus, of the pyloric portion, or stricture of the pyloric orifice, produce more or less dilatation of the whole organ. Its position may, under such circumstances, be changed, as well as its relation to the other organ enormously altered. In excessive contraction from cancer it is placed behind the ensiform cartilage, and presses the diaphragm into the thoracic cavity. When the cancer spreads from the stomach upon the mesocolon and colon it is drawn downward by the latter viscera, and then occupies a place immediately below the ensiform cartilage and above the navel. In excessive dilatations it may descend so low that the fundus either presses the transverse colon down into the pelvis or the greater portion occupies a position below the umbilicus and presses the smaller intestine downward and outward on each side.

The most peculiar displacement is produced by large-sized tumors in the lesser omentum and mesentery. These tumors occupy the smaller curvature, become firmly attached to the wall of the stomach, press it down and to the left, and cause the organ to occupy an almost vertical position. Under such circumstances it becomes enormously atrophied, reduced in size, and assumes the form of a large tumor. The compressed condition of the organ will prevent any vomiting from taking place.

Fig. 1.

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R

Fig. 2.

H.L.B

C

H.D.

F

S

Fig. 4.

Fig. 3.

F

Œ

O.C

D

P.E.

O.P.

R

G.E.

Fig. 5.

O.P.

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DISEASES OF THE ORGANS OF DIGESTION.

INFLAMMATION, HYPERAEMIA,

CONGESTION OF THE MUCOUS MEMBRANE OF THE INTESTINES.

Sec. VI. *Tab. IX.*

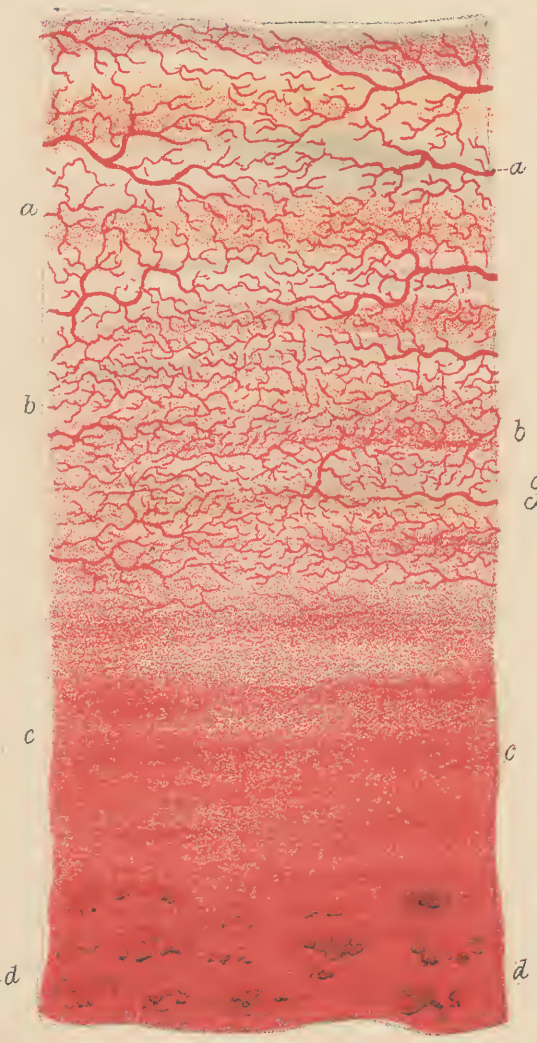

Fig. 1.

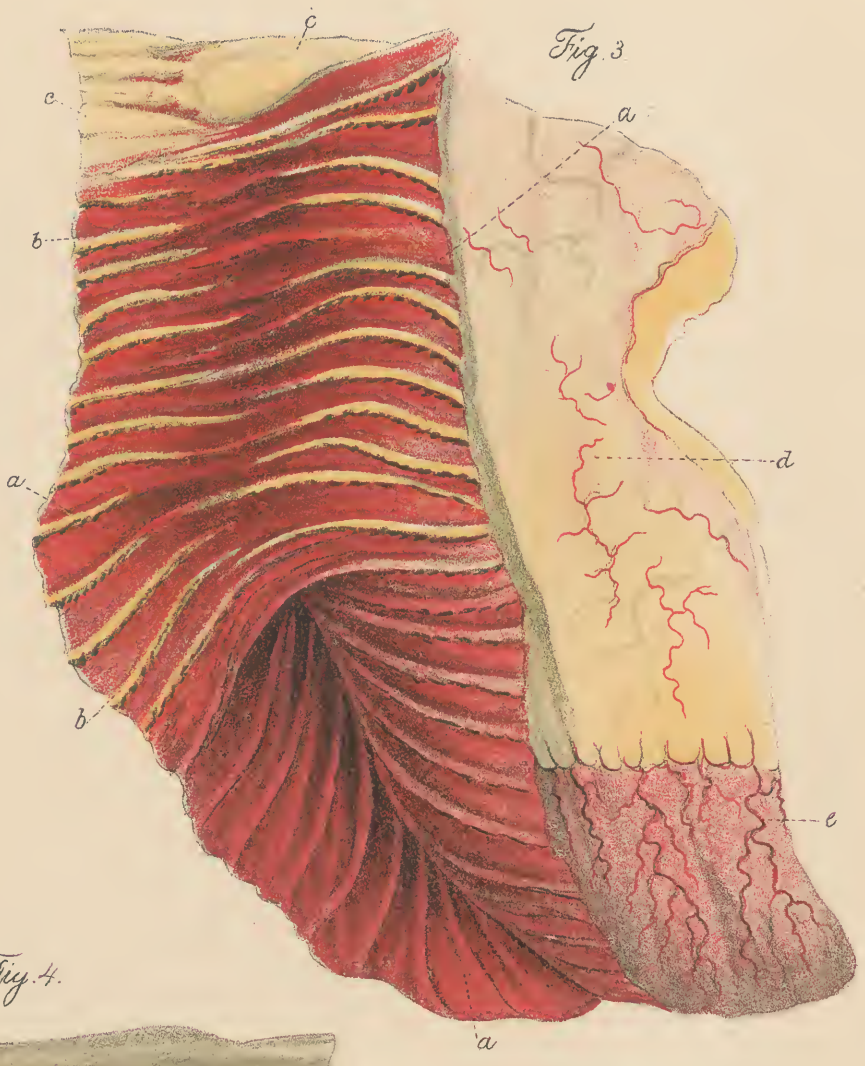

Fig. 3

Fig. 4.

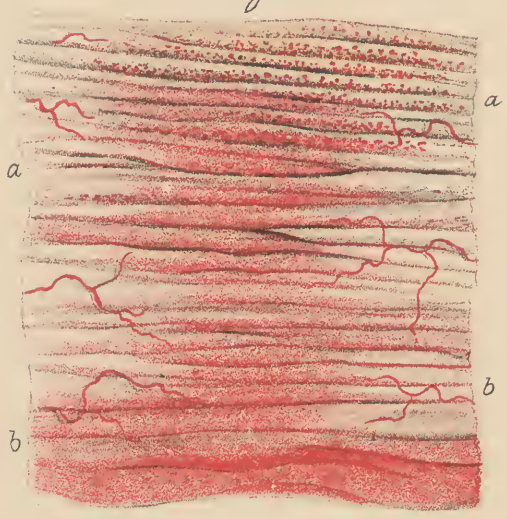

Fig. 2.

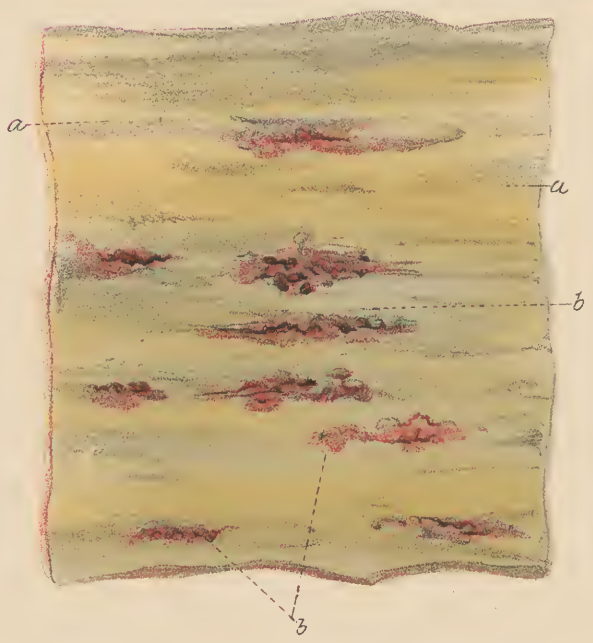

Fig. 6.

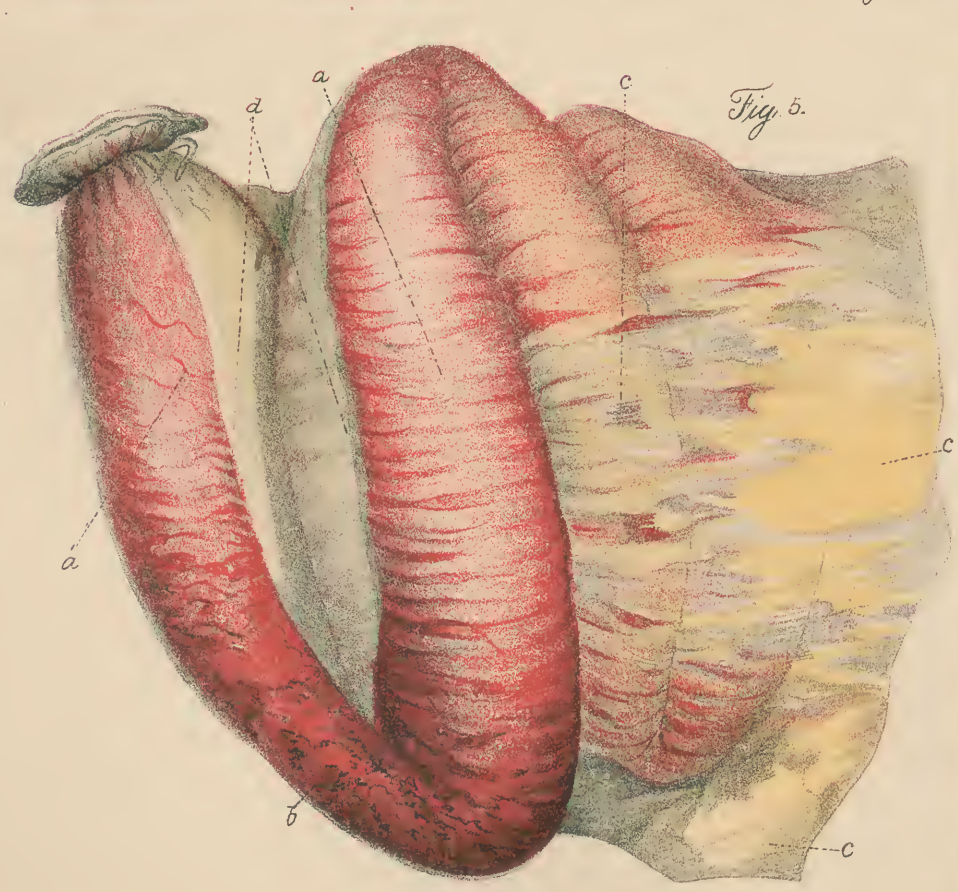

Fig. 5.

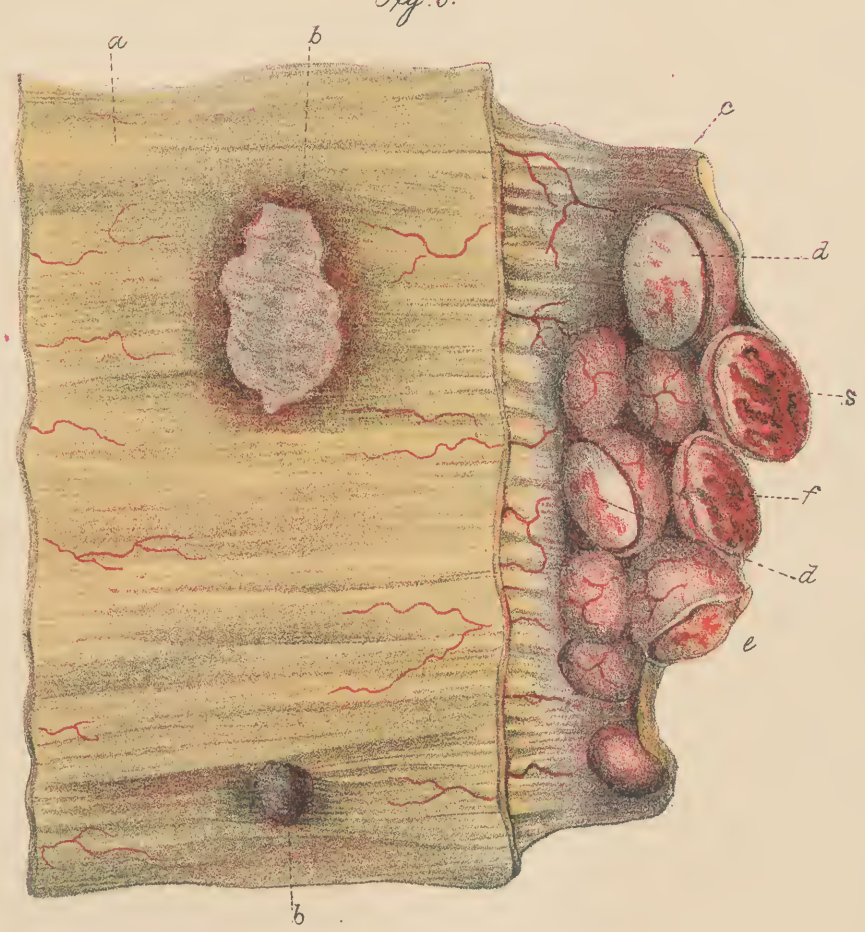

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TABLE IX.

Inflammations, Hyperæmia, Congestion, Ulceration of Mucous Membranes of the Intestines.

FIG. 1.— A portion of the small intestine showing the several forms of redness and vascularity of the inflamed mucous membrane (A A), ramiform injection, passing into the cappillariform (B B). The capillariform passing into the uniform (C C), and this passing into the spotted or hæmorrhagic (D D).

FIG. 2.—Punctiform redness (A A) of the villous structure of the ileum (B B), uniform redness in spots.

FIG. 3.— Mechanical congestion and inflammation of the ileum, in strangulated hernia (A A), the mucous membrane of a uniform deep red color (B B); projecting borders of the plicæ (C C); the mucous and sub-mucous tissues impregnated with pus (D), mechanical congestion of the external surface of the intestine (E), peritonitis.

FIG. 4.— Acute and chronic inflammation of the colon. The chronic inflammation is indicated by the slate-gray color (A A) (*sometimes, like in this figure, rather whitish gray*) of the mucous membrane. The acute attack, which has supervened upon the former, is seen by its red color, which is here in a hæmorrhagic or spotted form (B B).

FIG. 5.— Inflammation of the peritoneum, representing the several degrees of redness and vascularity accompanied by effusion of coagulable lymph (A A) folds of the small intestine along the contiguous border of which the inflammation is most severe, as it always begins in these situations and extends over the exposed surface of the intestine. At B the hæmorrhagic character is shown, where the blood has penetrated into the serous membrane, in the form of dark red spots. On the adjacent fold of the intestine (C C C) layers of coagulable lymph are found in the shape of pseudo-membranes. The inflammation in peritonitis usually begins along the contiguous margins of the neighboring folds of the intestine, and from thence extends in the direction where the blood vessels are distributed. Often it does not extend backward over the intestine toward the mesentery, the portion of the peritoneum between the margins of the intestine in this situation presenting its natural color as at (D) where the folds were separated to show this apearance. Hence it often happens that the anterior half presents all the characteristic appearances of acute inflammation, whilst the posterior half of the intestine is to all appearance healthy. (*R. Carswell.*)

FIG. 6.— Inflammation of the mesenteric glands accompanying ulceration of the intestine in typhoid fever, (A) portion of the small intestine laid open, (B b) ulceration of Peyers plaques (C Mesentery), (D D) glands enlarged and in the first stage of inflammation, (E F S) other glands presenting progressive stages of the disease and their terminations.

Mesenteric tumors alone do not change its shape, but alter its position according as they draw it to one side or to the other.

Tuberculous and carcinomatous tumors often draw the pylorus downward and completely cover it, causing it to occupy an almost vertical position. An enlarged spleen often draws the stomach to the left; it then occupies the hilum of the spleen, and is often covered by the latter.

Mechanism of the Smaller and Larger Intestines.

The intestinal mucous membrane adapts itself to the different surfaces as to the different functions, which are carried on in its several cavities. In cavities, which serve only as storing-places, or as ducts, there is found only a simple stratum of tough connective-tissue fibers facing the epithelium as a smooth, even layer, but sending fibrous prolongations from its nether surface into the apposing sub-mucous structure, and blending with it forms one tissue. But where the functions of the membrane are to secrete and to absorb, there it becomes a vast glandular structure, its free surface assuming enormous dimensions by forming innumerable folds, some projecting above it, some penetrating inwardly, thus representing an intricate wave-line in its horizontal projection. This multiplies the points of contact of the chyme and chyle with the absorbing surface. From the jejunum onward the inner intestinal surface is covered with various sized hair-like projections (*villi*), each containing in its center an absorbing lymphatic, and the connective-tissue layer between the vessels and the epithelium is of the kind of structure found in the stroma of lymph-glands. Solitary conglobated follicles and Peyers plaques or patches constitute additional absorbing arrangements. The latter constitute as it were the first station where the substances to be absorbed have to collect and through which they pass onward into the lymph circulation. When those substances contain any irritating material, they will at once affect the absorbing apparatus; hyperæmia, inflammation, and hyperplastic processes will be found going on foremost in those follicular structures. (Rindfleisch.) Like the stomach the other portions of the alimentary canal are not only supplied with abundant blood circulation, and subject to periodical hyperæmia during their functional activity, but also the peculiarity of vascular arrangement and the quantitive distribution of the arteries and veins render these organs fit for the double function of alimentation and limited respiration. During digestion a very extensive exchange of oxygen for carbonic acid is carried on not only between the oxygen of the air contained in the food and the blood, but also between that of the blood and the carbonic acid in the tissues. As in the stomach the additional oxygen respired increases the functional activity of the secretory apparatus, so in the intestine both the secretory and absorbing processes are thereby enhanced. For this purpose a description of the detail of the vascular distribution in these organs may not be out of place. *Frey* and *Gerlach*, to both of whom we owe the better acquaintance of the vascular arrangements in the intestines, describe them as follows: The arteries send up from the *nervea*, where they divide up in fine dendroid ramifications, parallel branchlets into the glands. In their ascent between the glands they still further sub-divide and anastomose by very numerous inosculating vessels, thus surrounding and covering the glands with a dense capillary net-work. From around the orifices there arise larger vessels, which, sub-dividing and forming a vascular layer in the interglandular parts, send up vascular loops into the villi.

From the superficial part of the mucous membrane the veins are formed from intricate capillary net-works. These veins quickly increase in caliber, penetrate into the deeper layers, and pass hence without taking up any branches from the glandular walls. (Henle.) The finer the membraneous layer covering such a vascular stratum the more fit is it to perform the function of osmosis and absorption; especially the more lymphatics are distributed within such vascular layer the more is absorption effected. Pressure produced upon blood capillaries favors exudation and diminishes the quantity of liquid in the vessels, but upon lymphatics pressure has a contrary effect. It increases absorption, for, as their very numerous valves prevent their contents from passing backward, the pressure necessarily drives the lymph forward toward the heart. The quality of this highly permeable membrane, which adapts it to osmotic function, renders it peculiarly subject to extensive infiltration, especially in that portion of the intestines, where it has a very uneven surface and is provided with many sacculated elevations and depressions, having yielding, spongy sub-mucous structure. On the slightest occasion can hyperæmia produce infiltration into the intestinal wall.

The Duodenum.

Functionally the duodenum may be considered as forming part of the stomach, for here also is digestion carried on, especially of such substances which have not undergone sufficient change to enter into the current of lymph. Although not so voluminous as that of the stomach the mucous membrane in this part of the intestine has those extensive duplicatures (*valvulae conniventes*), folds of the functional layer, placed nearly perpendicular to the axis of the tube and almost parallel to each other. Both the muscular and mucous tunics are direct continuations of those of the stomach, and the latter tunic is readily movable upon its sub-mucous structure. The secretion of the gastric end of the duodenum is like that of the stomach, of a distinctly acid reaction (Richet), and is subject to the same class of morbid changes as the structures of the stomach (circular ulcers, self-digestion by its acid secretion, etc.)

From the jejunum onward the alimentary tract has more the character of an absorbing than of a digesting organ. From the colon down no digestion at all and but limited absorption takes place. Water, some saline and fatty substances, non-peptonized albumen, and some peptones are absorbed there but extremely slow, and more by simple transudation than by actual physiological action. (*M. Markwald, Virch. Arch., Vol. LXIV, p.* 505; *Latschenberger, Czerny, Virch. Arch., Vol. LIX, p.* 161. Also, *Braune, Funke, Thiry, Quinke, Eichhorst, Voit, Bauer.*) What digestion is carried on in the smaller intestine is only between the pylorus and the coecum by the enteric juice which *Leven* and *Vela* described as an opalescent watery liquid of a marked alkaline reaction; of course, bile and pancreatic juice are comprised within its composition. Absorption does not take place in all parts alike, even of the smaller intestines; the difference in absorbing power exists in the structural arrangement of the epithelium in the several parts of the mucous membrane, as *Lannois* and *Lepine* have proven by their experiments upon the individual portions of the alimentary canal. (*Compt. rendues, de la soc. de biologie, p.* 92.)

Pathological Anatomy of the Intestines.

The morbid anatomical changes in the intestine below the stomach, although resembling by many features those of morbidly changed mucous membrane generally and that of the stomach particularly, differ as the different portions of the tract have different anatomical arrangements of the essentially same structural elements, as specific alterations will necessarily follow specifically differential morbid conditions. It is not a matter of indifference to the functional activity of the stomach that its mucous membrane, and especially the epithelial layer, is far more voluminous than that of the Ileum, Colon, or even the duodenum or not; that its sub-mucous tissue is more elastic, that its muscular tissue is vastly more developed and in greater proportion, that its arteries are provided with much more abundant collateral circulation than any of the middle or lower intestines, or not. If not so well supplied with absorbent arrangements on its free surface as the small intestines, its function being more a digestive than absorbent, the digestive liquid of the stomach being highly acid, its mucous glands are exceedingly developed and wonderfully fit for pouring out thick, viscid mucous to protect itself against its own gastric juice, whilst its epithelium, of very speedy growth, readily softens and becomes dissolved to form digestive *zymogen*, and as is readily replaced by new epithelial structure to maintain the histological integrity of the organ. The duodenum, less voluminously developed, both as to its mucous and other structures, is provided with more lymphatics and lymph-glands, with a

TABLE X.

Hyperplastic Formations, Infiltrations, Fibroid, Vascular and Membraneous Neoplasmata; Hypertrophy and Atrophy of the several Tunics of the small Intestines.

FIGS. 1, 2.—A small pyriform polypus attached by a peduncle to the wall of the abdomen and covered by a serous membrane derived from the peritoneum, (A) tumor, (B B B) small vessels ramifying under the peritoneum and passing into the tumor.

FIG. 2.—Section of the neoplasm showing its interior nucleus (C) to be rather orange color, whilst externally it is of a bright red (B).

FIG. 3.—Neoplastic membranes and newly-formed vessels in those adhesions uniting the intestines with each other and with the parieties of the abdomen, (A A) portion of the intestine, (B B) portion of the abdominal wall, (C C) adhesions, in which the blood vessels are seen.

FIG. 4.—Cicatrizations of ulcers of the agminated glands, in follicular catarrh, (A b c) agminated glands, (D e) cicatrices consisting of simple mucous tissue, (F G) two small ulcers incompletely cicatrized, their edges still sharp, (H) an ulcer undergoing the process of reorganization.

FIG. 5.—Imperfect cicatrization of an ulcer of the small intestine, which has destroyed the muscular coat around the whole circumference of the tube. It has been replaced by the contractile fibrous tissue (A), which has here a stellated arrangement, and has, by its contraction, narrowed the intestinal cavity (B B) considerably.

FIG. 6.—A remarkable example of stricture of the small intestine from the presence of the same tissue in a cicatrix occupying the entire caliber of the intestine. (A a) upper extremity of the ileum; (B b) lower extremity. (C c) fibrous tissue stretching across the intestine in the form of bands, leaving small openings (D D) between them, and through which but small quantities of liquid fæces could pass.

FIG. 7.—A portion of the ileum with a croupous deposit upon its mucous membrane, in croupous enteritis.

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greater number of veins, although smaller-sized, than the former viscus, etc., performs supplementary digestion; its digestive liquid is *mainly* alkaline, does not require the great quantity of blood serum to neutralize it as the stomach; its arteries are very numerous but far smaller, and supply oxygen to its tissues. The biliary substances, necessarily more or less absorbed in the wall of this intestine, cause a slower flow of its blood and a greater stagnation in its vessels, which, by their being enveloped in the mesenteric folds, are already hemmed very much in their circulatory function. Yet this slow circulation permits a great quantity of serum to enter the cavity and dissolve its digesting contents, preparatory to their absorption by the chyliferous vessels. The arterial pressure is usually low, but is subject to nice regulation by the very numerous vasomotor fibers derived from many sources of the sympathetic plexuses. After digestion has taken place in its several cavities the intestine is only supplied with sufficient blood for its own nutrition, and is in this condition usually of a pale or very light pink color (*in infants*).

Inflammation, Hyperæmia of the Intestines.

The line of demarcation between the physiological and pathological hyperæmia is very hard to draw in the intestine. Post-mortem appearances are the most deceptive in regard to the color or the quantity of the blood in the organ. The most intense state of hyperæmia during life may show the intestine of nearly pale color after death. On the other hand, imbibition of its tissues with blood takes place so easily after death, and even during prolonged death agony, that intense redness and great injection of the intestinal structure can be found after death, where, during life, it was perfectly normal. Still, hyperæmia, during life, seldom fails to leave some traces behind, if it has existed for any length of time, and has caused the vessels to undergo such changes which can be revealed by the microscope. Sometimes static congestion even impresses upon the endothelial stratum of the vessels, and upon the epithelial layer of the mucous membrane such distinct marks that, notwithstanding the paleness of the tissue, they can be recognized without great difficulty.

Of the different forms of inflammation affecting the intestines the catarrhal is the most frequent (*catarrhal enteritis*). The inflammatory process furnishes sometimes mucous, sometimes serous, sometimes puriform, and often mixed secretions. In the colon the quantity of mucus produced by the inflamed epithelium is sometimes enormous, and is carried off as very copious mucus discharges.

By far the greatest number of catarral inflammations of the tract are of a transient character, and as a rule terminate favorably, yet sometimes leave behind quite noticeable anatomical alterations. Even in moderate intestinal catarrh the connective tissue between Lieberkuhn's Krypts become infiltrated with plasma and lymphoid corpuscles. A portion of the hypersecreting epithelial cells are thereby loosened from their sub-stratum, and are gradually destroyed. When the inflammatory process reaches a high degree, and persists for any length of time, there occurs extensive epithelial exfoliation without any reformation of new structures in their places, and the intestine becomes atrophic. Light grades of atrophy are not readily recognizable by the naked eye. (Possibly the very thin and glossy appearance might indicate it.) But even a low magnifying power will show its atrophic condition by the glandular layer being much thinner than normal, sometimes reduced to one-half, or even a third, of its usual thickness. The same alteration is found in the villous stratum of the smaller intestine. Ulcerative processes cause still higher degrees of atrophy of the intestinal wall, for not only is the epithelium obliterated but the other structures are gradually destroyed in succession. (*Sec. VI, Tabs. V, VI, VII,* show the various degrees of successive destruction of the intestinal walls by inflammatory and ulcerative processes.) The mucous membrane presents, under such diseased circumstances, its surface covered either with mucus, pus, or whitish or yellowish flakes, consisting of granules, or shreds of the mortified portions of the infiltrated upper portions of the glandular stratum. (*Sec. VI, Tab. XIII, Figs.* 1, 2, 3.) Ulcerative inflammation can, in a very short time, produce not only a loss of the upper layer of the mucous membrane but also of the whole glandular stratum, leaving upon the muscular surface only a very thin pellicle of highly nuclear connective tissue. (*Sec. VI, Tab. XIV, Figs.* 1, 2, 3, 4.) Ordinarily the muscular tissue is but little altered in catarrhal inflammations. Its fibers are very seldom atrophied, or fatty degenerated. The sub-mucous is usually also but very little altered, but in ulceration of the mucous coat it is often strongly infiltrated with lymphoid cells, and somewhat thickened and indurated. The follicles in ordinary catarrhal inflammation are, with the exception of a slight enlargement, but little altered; but in the ulcerative form they are usually most involved, and very soon destroyed by suppurative process. (*Sec. VI, Tab. XIV, Figs.* 3, 4, 5, *etc.*) (*Follicular catarrh.*) Upon the membrane there are formed ulcerating depressions of greater or lesser extent in the place of the follicles (*follicular ulcers*).

Atrophies following catarrhal inflammations are mostly found in the colon, and especially in and around the coecum. Nothnagel found eighty per cent. of enteric atrophy of adults to be only in the coecum. Next to this, in frequency of this affection, is the ascending colon. The higher above these the rarer is this affection found in them. In small children enteric atrophy is found in any portion of the intestine after acute and chronic catarrh. The muscular tissue suffers but little change in catarrhal trouble except when associated with static hyperæmia, and then it is rather hypertrophied than atrophic. (*Nothnagel.*) Although little inclined to much alteration, the muscular tissue undergoes fatty degeneration in consumptive diseases. (*Wagner.*) Occasionally there is found congenital atrophy of the intestinal muscular tunic. (*Nothnagel.*)

In chronic catarrh there may be found atrophy and hypertrophy side by side in this tissue, hyperplastic formations, either in the shape of mere indurations of the sub-mucous and muscular structures, or in the form of excrescences or polypi upon the mucous surface. They resemble, in form and structure, those described among the diseases of the stomach, as, *etat mamelonnee*, or *gastric polyposis.*

Diphtheritic lesions are often found in the lower portions of the smaller and upper portions of the larger intestines, especially associated with ulcerative catarrh, in the form of minute furfuric scales upon the very red and exceedingly swollen surfaces of the mucous tissue. Sometimes in larger plaques. This most frequently exists in specific inflammations and infectious diseases. Extensive fibrinous exudates are very rare, yet it sometimes happens that a large portion of the intestine is enormously swollen, intensely red, and hyperæmic, and covered with a fibroid membrane. Fibrinous exudations of small dimensions are very often found in catarrhal ulceration by the side of diphtheritic patches. (*Wagner, Kundrat, Gerhardt, Woodward, Damaschino, Kussmaul, Maier,* and *Schwark.*)

Histological Changes of the Intestinal Structures.

They are as follows:

1. *Desquamative Catarrh.*—In sudden diarrhœa of even a healthy person, in which there are very copious, watery discharges from the bowels, mixed with jelly-like or opaque mucous masses, in the form of shreds or membranes, the microscope shows a vast number of epithelial cells in the serous and saline liquids of the stools. Occasionally the cells form cohesive membranes. These are the exfoliated epithelium of the mucous membrane. Most of the cells are bloated and enlarged, their nuclei are dimly outlined, a little more refractive than their protoplasm, which is changed into a finely granular mass. Occasionally the whole lining of a krypt is found among the cells, forming a coherent *circlet* of cells in radiary arrangement. In chronic catarrh, the cells are purplish or deep red, unusually granular, with very indistinct nuclei, and contain many fat molecules not derived from the intestine but from degeneration of its own protoplasm.

2. In light follicular catarrh neither the microscope nor the naked eye is able to discover much change in the mucous membrane; but in graver forms of this inflammation, the alterations in the solitary glands and *Peyers* patches are very characteristic. According to the intensity or duration of the inflammatory process, the follicles project more or less above the surface of the mucous membrane, and are usually thus recognized by the naked eye. The redness of the otherwise normal mucous membrane remains, even after death. The follicular enlargement is at first produced by excessive hyperæmia, subsequently by cellular infiltration. In the more advanced stages of the disease, the interfollicular connective tissue becomes similarly infiltrated to such an extent that it will destroy the follicular walls and produce communication between the several follicles, giving rise to formation of abscesses in the communicating sinuses, and ultimately ulceration within the plaques. The outer surface will thereby assume a reticulated appearance; or extensive portions of the mucous surface will exfoliate in a body by the undermining abscesses. The capillaries in these portions of the

HYPERPLASIA, PURIFORM AND FIBROID EXUDATIONS. INFILTRATIONS WITH BLOOD AND WITH PUS.

Sec. VI. *Tab. X.*

Fig. 1.

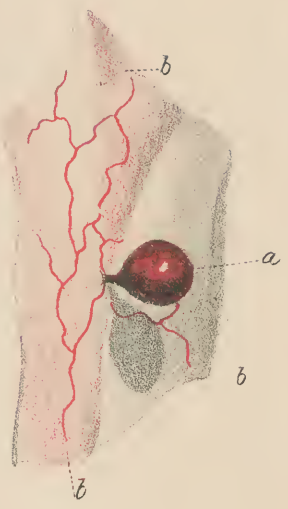


Fig. 2.

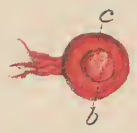


Fig. 3.

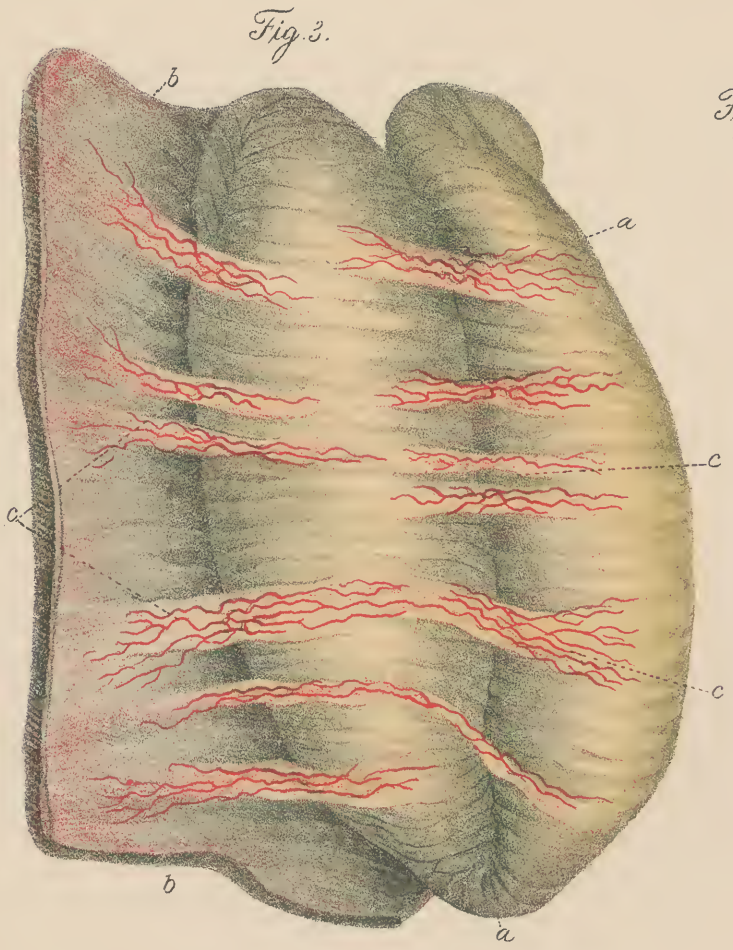


Fig. 7.

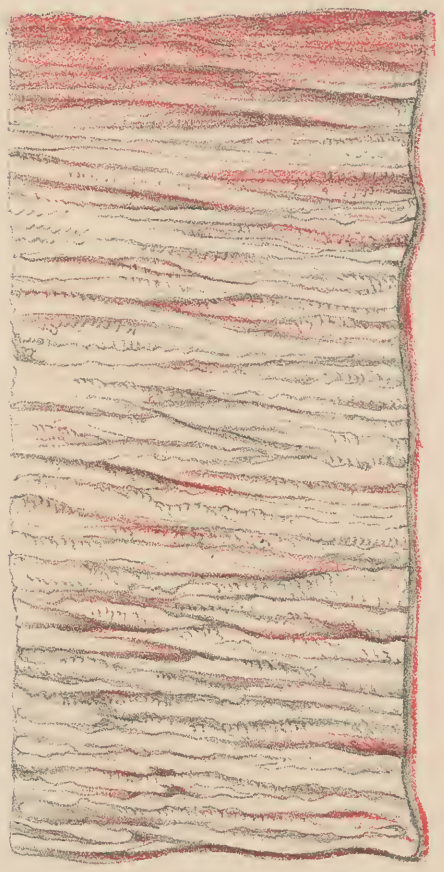


Fig. 4.

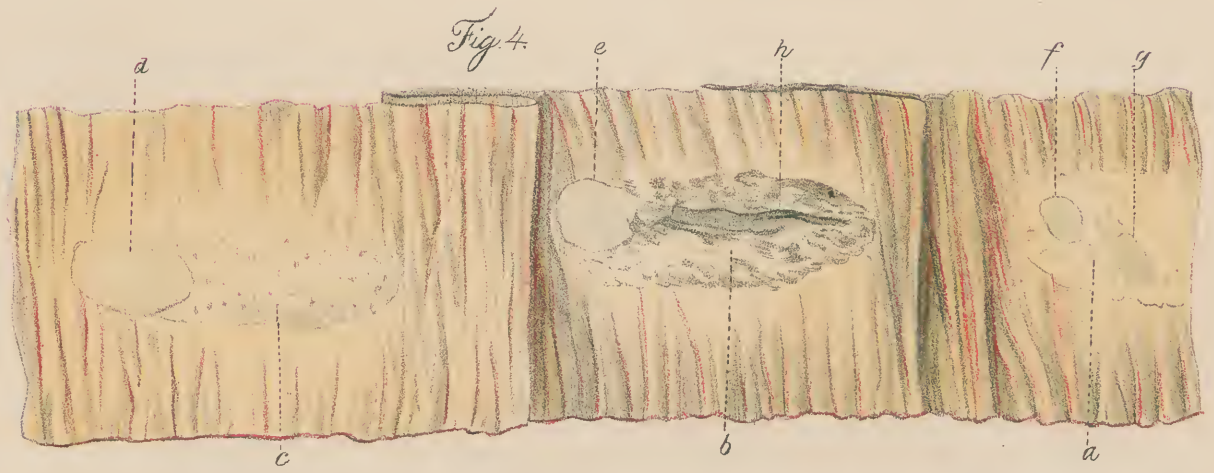


Fig. 6.

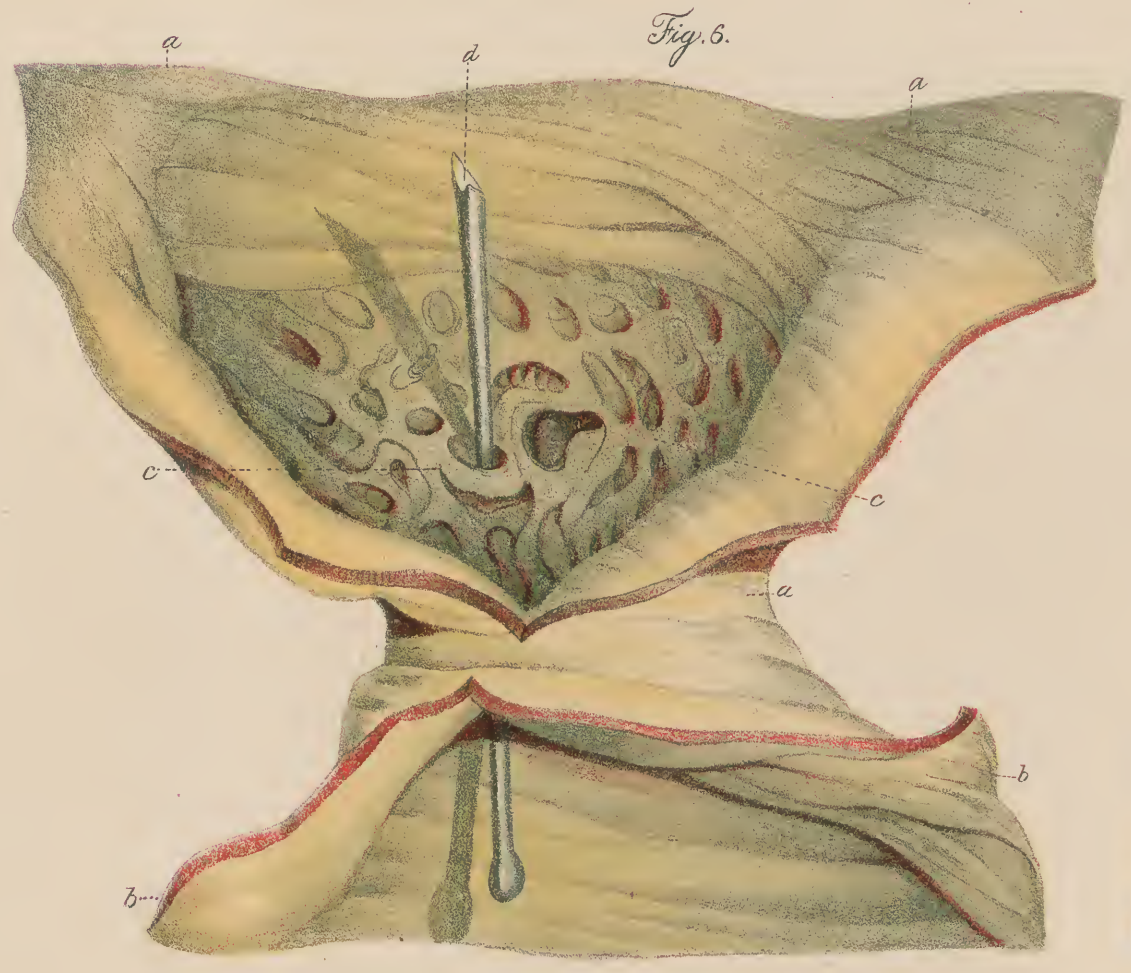
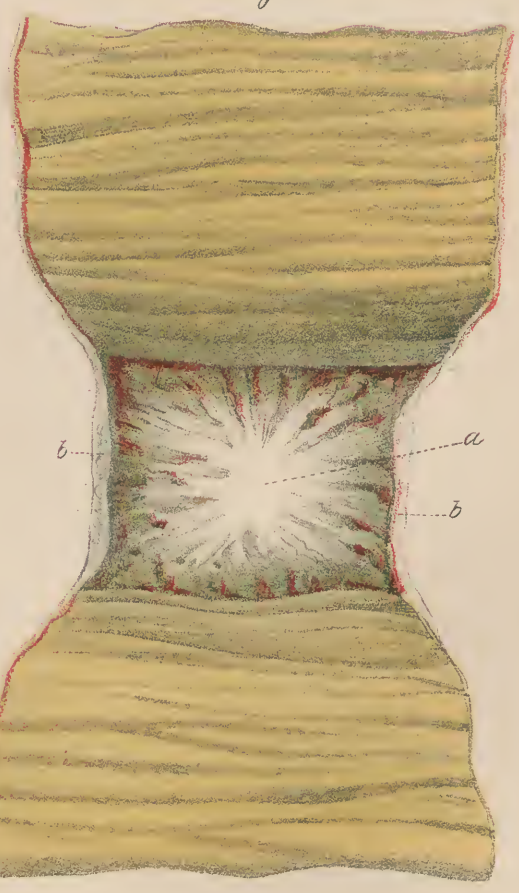


Fig. 5.



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HAEMORRHAGES. HAEMORRHAGIC ULCERATIONS HAEMORRHAGIC INFARCT.

Sec. VI. *Tab. XI.*

Fig. 1.

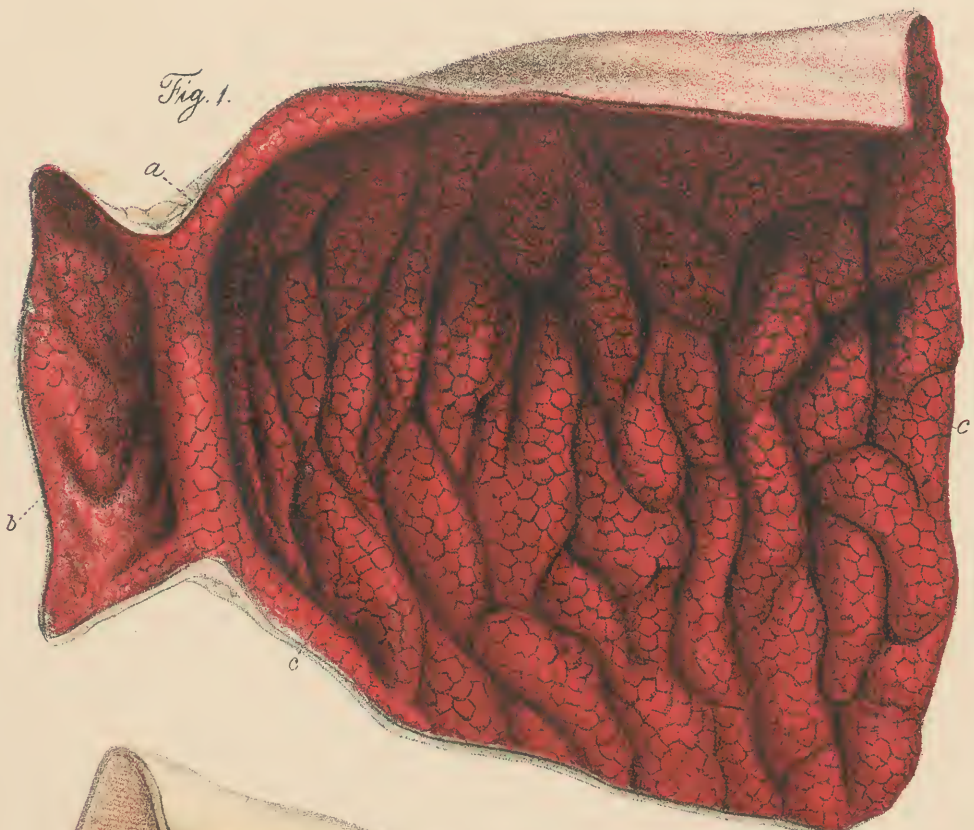

Fig. 2.

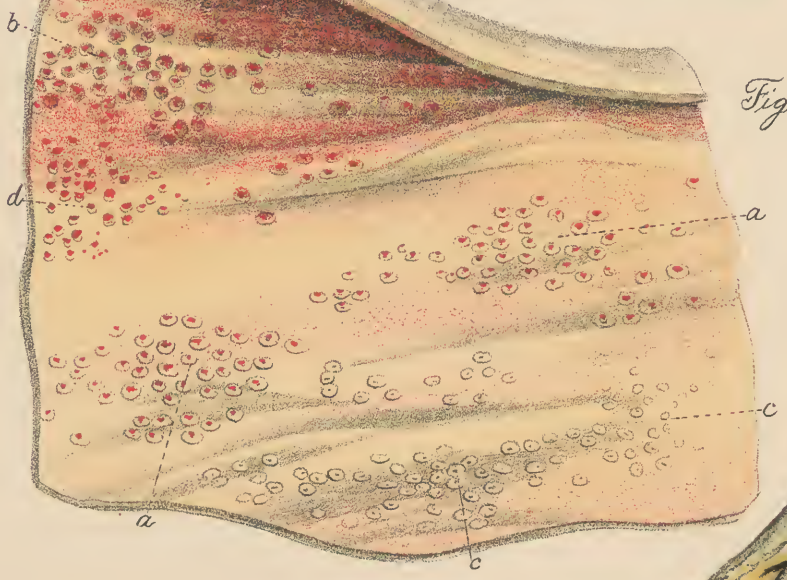

Fig. 4.

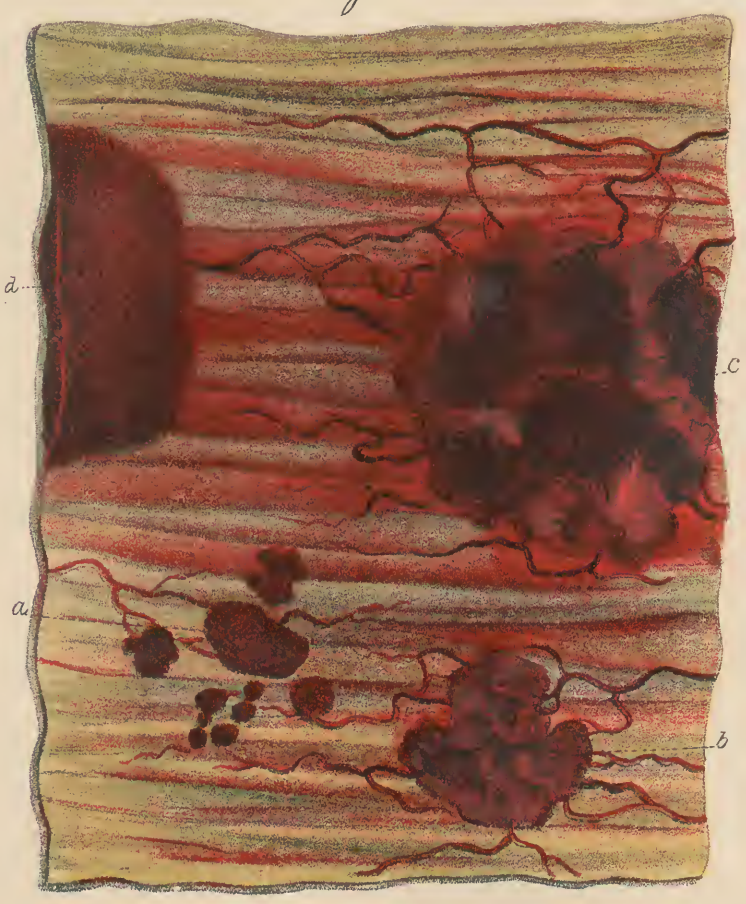

Fig. 5

Fig. 3.

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Table XI.

Fig. 1.—*Hæmorrhage of the Mucous Membrane of the Stomach, from Mechanical Obstruction to the Return of the Blood, situated in the Heart.*

(A) pyloric portion; (B) commencement of duodenum; (C) the mucous membrane thrown into folds of a uniform redness and presenting a granular appearance from enlargement of its follicles.

Fig. 2.—*Hæmorrhage of the Stomach in Follicular Ulceration.* (A A) enlarged follicles, its bodies are pale, but have a red central orifice; (B) other enlarged follicles, but differing from the above in their bodies being also red; (C) follicles pale throughout, with a central depression; (D) petechiæ of the mucous membrane accompanying this form of hæmorrhage.

Fig. 3.—*Hæmorrhage of the Duodenum, from Ulceration of its Mucous Membrane.* (A) duodenum; (B) pyloric portion of the stomach; (C) ragged ulcers of the membrane penetrating to the sub-mucous tissue and surrounded by elevated borders of the mucous structure.

The surfaces of the ulcers are covered with dark blood coagula, and the membrane presents a deep brownish red color from imbibition. (D) a small ulcer of the follicles; (E E) two small circular ulcers in the pyloric portion of the stomach.

Fig. 4.—Hæmorrhagic *infarct*, in the mucous membrane of the colon, from embolic occlusion of the superior mesenteric artery. The hæmorrhagic patches (a a) present a uniform dull red color, with slight congestion only, the effused blood being confined to the mucous membrane alone. At (b and c) the extravasation is more extensive, and occupies the mucous as well as the sub-mucous tissue, and is associated with extensive venous stasis and varicose dilatations of those vessels, presenting also here and there deep red and black blood coagula. The infarct (D) is situated between the mucous and sub-mucous layers. A portion of the first is thereby raised as a flat spherical elevation.

Fig. 5.—*Hæmorrhagic Ulceration in the Duodenum.* The mucous and serous surface of the intestine is here shown. The valvulæ conniventes of the duodenum (P D) are all covered with fibroid and puriform matter, with here and there black gangrenous points between them. The mucous tissue is swollen and thickened. The serous surface presents many hæmorrhagic spots, some of great extent. Hæmorrhagic ulceration and destruction of portions of the serous membrane (H U), showing the denuded muscular coat (M). The borders of the ulcers are only slightly raised (as is usual in perforation of the serous membrane by ulcerative destructions), in some places not at all. There is extensive infiltration of blood and pus in the tissue (H) surrounding the ulcers. The serous membrane is also impregnated with blood pigment in those localities.

tissue are enormously injected, and many small hæmorrhages appear upon the surface and in the parenchyma. These hæmorrhages take place even in mitigated cases of the inflammation, as the subsequent pigmentations upon the structure readily prove. In a case of a follicular ulcer confined to the colon (of a child described by Thierfelder), the intestine was much contracted; its mucous membrane covered with abundant mucus, colored with biliary pigment, and showed very numerous miliary hæmorrhages. The membrane was moderately swelled and much infiltrated with lymphoid cells into the inter-glandular tissue. The glands were not compressed, but seemed rather dilated and filled with mucus. The hyperæmia, which must have existed during life, had completely disappeared. The sub-mucous tissue was œdomatous, but otherwise slightly changed. The vessels were somewhat injected, and the muscular and serous tunics but little altered.

3. In the stomach and the duodenum suppurative inflammation is frequent; in the intestines it is very rare. Its clinical character is that of an idiopathic fever, very analogous, in its symptoms, to phlegmonous and erysipelatous inflammation of the skin. The phlegmonous process may extend over the whole intestinal tract, in all its layers, and lead to exceedingly extensive ulceration. In the superficial layers there is but little alteration, except some collection of few pus-corpuscles around the little dilated capillaries. In the mucous tissue proper, and in the interstitial structures between the tubular glands, there is enormous infiltration of pus, which compresses the glands. Extensive extravasates of blood are pressed between the tissues, and the epithelium loses its characteristic cylindrical form from this very pressure.

4. Amyloid degeneration in the intestinal vessels is as frequent as in those of other viscera of the abdomen, only here they are not so prominent as in the others. The walls of the smallest arteries and capillaries are usually first affected; the muscles and the epithelium become subsequently involved. The altered tissues have a glassy, translucent appearance, and turn purplish or brownish-red when treated with iodine and some sulphuric acid. The affected parts lose their elasticity; they close up the vascular cavity, and thereby produce local anæmia of the tissues and their subsequent death.

The inflammations of the different portions of the intestine have been denominated according to the name of the affected part, viz:

Duodenitis.—Inflammation of the duodenum usually occurs simultaneously with that of the stomach. Hyperplastic formations in that cavity often close up the orifice and portion of the common choledic duct; especially the inflammation extends upward into the bile ducts. By this closure, although even temporary, there is produced catarrhal icterus, which sometimes assumes a very grave form of blood-poisoning. As in the stomach so may also be found circular ulcers in the duodenum.

Ileitis.—Inflammation in this portion of the tract is generally characterized by swelling of the solitary glands and aggregated follicles. (*Follicular catarrh.*) The solitary follicles appear as reddish or grayish-white prominent nodules; the aggregated as grayish or reddish-gray patches or flat ridges, with many small depressions or pits on top of these. Destruction of the follicles produces follicular ulcers.

Typhlitis and *Perityphlitis* are names given to inflammation of the vermiform appendix and its adjacent tissues. This portion of the intestine is, from the nature of its position and structural arrangement, much subject to retention of many substances passing through the intestine. Some of these cause very great irritation in the cul de sac. The inflamed tissue and the substances causing the inflammation often become incrustated with earthy phosphates and carbonates. (*Koproliths.*) These deposits may extend upon all the surrounding tissues and thereby produce ulceration, followed by mortification and perforation of the intestine. When these foreign substances produce inflammation confined only to the cavity of the appendix, and the exudate is not very extensive, the cavity may close up by neoplastic tissue. But when perforation of its wall takes place before the cavity can close up, its contents are emptied into the peritoneal spaces, and fatal peritonitis is the consequence. It happens sometimes that excrements are retained in the appendicular cavity whilst its orifice in front is closed up by neoplastic matter. Abscesses are then formed which may perforate the walls and discharge either into an intestine or externally into the peritoneum. Sometimes the whole cavity becomes thus filled up with newly-formed hyperplastic material, or it may only become partially filled, and the still open cavity will gradually fill more and more with secretion from still functioning mucus membrane and dilate to an enormous extent. Under other circumstances the appendix becomes atrophic and is almost obliterated, or becomes attached by adhesive inflammation to some portion or other of its adjacent tissue. Inflammation into the appendicular cavity is often extended from the ileum or the cœcum, or from both. Especially in tuberculosis of the lung and tubercular infiltration in the mucus membrane of the cœcum is the appendicular organ highly deformed by extensive ulceration and sometimes almost obliterated. In (Sec. VI, Table XVI) a case reported by *Richard Bright*, in his celebrated *Report of Medical Cases* (Fig. 1), is given an excellent representation of ulceration of the cœcum and almost total obliteration of the appendix found on post-mortem in a consumptive patient of Guy's Hospital, London. Inflammatory, typhoid, and tubercular infiltrations into the appendix are often very dangerous, and sometimes even prove fatal complications in those diseases.

Inflammation of the colon is a very frequent disease (*Colitis*). Its several peculiar forms have been described above. Its causes are many and various. Impaction of fæces, septic infiltrations, or specific infections, and sometimes poisons are usually the inciting causes of those diseases and lesions.

Inflammation of the Rectum—Proctitis.

In many respects, both in regard to symptoms and etiology, the affections of the rectum resemble those of the vermiform appendix. Presence of foreign bodies is the most frequent cause of its several lesions and derangements. Venous stasis is usually the immediate cause of inflammatory processes therein. Ulcerations, fibroid hyperplasia, thickening of the intestinal mucous membrane, and vegetations upon its surface are the most frequent results of those inflammations. Its surface readily becomes covered with puriform mucus. When the inflammation or ulceration penetrates into the deeper strata of the rectal wall the adjacent soft connective tissue becomes infiltrated and hyperplastic; or there are formed perirectal abscesses and suppurating gangrenous pockets. (*Periproctitis.*) Sinuses and canals are then formed from the mucous and sub-mucous structures into the adjoining tissue; so-called *incomplete fistulæ*. When the sinus and abscess open externally they form *complete external fistulæ*. When the external orifice communicates with the rectum by a canal, *complete rectal fistulæ* is then produced. The sinus may then be either lined with a layer of secreting epithelium or with a pyogenic membrane. Recto-vesicular and recto-vaginal fistulæ are also thus formed. Syphilis, tuberculosis, dysentery, and other infectious matters, carcinoma or ulcerations, are all very liable to produce periproctitis. Puerperal sepsis, pyæmia, typhoid, and rheumatic infections very often produce periproctitis without preceding ulcerations.

Intestinal Inflammations produced by Specific Infections.

Epidemic, endemic, and sporadic dysentery constitute an inflammatory affection of the colon. The specific infectious substance or agency has not yet been definitely ascertained. It is undoubtedly one or another of the many forms of schizomycetes found in many localities in the ground and in water. The character of anatomical alterations in dysentery is very similar to those found in the intestinal affections by a non-specific agency. This is the reason why its morbid anatomy has as yet not furnished any peculiar diagnostic phenomena of dysentery. The intensity of the dysenteric process and its extent differ very much under various circumstances; the whole colon or only a small part may become involved, from the rectum, the signoid flexure, ilio-cœcal valve, or even

TABLE XII.

Acute and Chronic Ulceration of the Intestines, with subsequent Gangrene and Perforations.

FIG. 1 presents a portion of the ileum laid open, the solitary and agminated follicles of which were the seat of inflammation, ulcerated or in a state of sphacelus. Several of the solitary follicles (a a) are seen surrounded by an inflamed elevated border, inclosing a yellowish gray slough, formed of the sub-mucous in some, from this and the muscular tissue in others. Several of the aggregated were in a similar state, and one of these had entirely sloughed and disappeared, together with the muscular and peritoneal strata, thus giving rise to perforation of the intestine (B) to the extent of an inch and a half in length and half an inch in breadth. (C) sharp edges of the perforation. (D) solitary follicles enlarged.

FIG. 2.—Perforation of the ileum, from sloughing of *Peyer's plaque*, in chronic follicular inflammation. (A) portion of the sphacelus still adhering to the edge of the perforation. (B) raised border of pink color. (C D) agminated follicles in a state of chronic inflammation.

FIG. 3.—Very extensive perforation succeeding chronic follicular catarrh. (A) sharp edge of the perforated structures. (B) portion of the plaque still left on the mucous surface. (C) a plaque in a state of inflammatory ulceration.

FIG. 4.—Hæmorrhage and perforation from chronic tubercular ulceration. (A) raised border of the ulcer infiltrated with tuberculous matter. (b c) sub-mucous muscular coat in the same condition. (C) sphacelus of peritoneal tissue. (D) border of another ulcer. (E) muscular coat. (F) serous membrane. (G) perforation.

FIG. 5.—External appearance of sloughing of the peritoneum in tubercular ulceration. (A) a probe passed through the perforated intestine. (B) center of the slough.

FIG. 6.—*Periproctitis and Phlebothromboses.* There is dilatation of the veins of the rectum and very extensive hyperplasia of the mucous and sub-mucous tissues, (A A) present venous stases, thrombotic condition of the smaller veins, and infiltration in the loose connective tissue. (B) a pendulous tumor, vascularized and subsequently transformed into a spongy erectile growth. (b b) other tumors of the same kind. (d d) varicose condition of the veins showing a partly red and partly white thrombus. (E) other veins varicose. (F G) highly dilated and tortuous veins.

FIG. 7 represents the internal surface of a portion of a colon affected with pale inflammatory softening of the mucous membrane, and its follicles. (A A) enlarged follicles and dilated vessels. (b b) circular patches formed by destruction of the follicles, its mucous covering still adherent to their walls. (C C) portion of sub-mucous structure denuded by destruction of the mucous surface, part white, part grayish blue. (D D) remnants of the mucous membrane left in ragged shreds.

beyond it, into the smaller intestine, the inflammation may spread with different intensity in different localities. In recent cases the mucous membrane is intensely congested and swollen, often filled with hæmorrhages, its surface covered with albuminous liquid usually mixed with bloody strings. The secretion assumes, very early in the disease, a mucus character, or one almost totally bloody. The scaly furfuric deposits (*Tab. X, Sec. VI, Fig.* 7) denote partial death of some of its surface tissue. (See also *Sec. VI, Tab. XIII, Figs.* 1, 2.) Denudation of the surface following such a condition will soon become readily visible to the naked eye. The disease has either a catarrhal or a diphtheritic character, or sometimes both. In the lighter forms the decay of the tissue is very superficial. In the graver forms there is found extensive destruction of the glandular layer only or of the whole mucous membrane. This membrane becomes an opaque granular mass, in which the form of the structural elements and their arrangements are totally obliterated. The decay of the superficial portions is usually not very extensive, and usually confined to the edges of the mucous folds. They have a grayish-white, gray or black color, whilst the still living tissue is of an intensely red or a grayish-red color. Upon the inter-mediary structure there is sometimes found a deposit of very fine bran-like flakes—white or of grayish-white, or even brownish—part of which are still adherent to and part detached from the surface. Larger flakes than the ones described very seldom exist. Below the mortified portions the tissues are very much infiltrated with lymphoid cells. Both the sub-mucous and the muscular tissue become also infiltrated. The lymph-follicles are distinctly infiltrated and soon become ulcerated.

When the quantity of pus is very great the mucous tissue may become undermined, and detached in large flakes, and the denuded tissue very readily ulcerates. Such exfoliation and subsequent ulceration may be confined to very small spots, or to very large portions of the surface. At any stage of inflammation its processes may stop and repair may begin. Of course, the less extensive the injury the more favorable are the prospects for complete recovery. Yet, even the most mitigated ulcerative conditions leave behind an atrophic state of the mucous membrane. Extensive and severe ulcerations are followed by extensive cicatrizations. Extensive destruction and long-protracted inflammations are succeeded by permanent and extensive alterations. The glandular stratum is then permanently obliterated, the deeper layers become tough and indurated, the connective tissue hypertrophic. There is formed more or less stricture of the cavity. The narrowing may become so excessive that mucus can be secreted by only a very limited portion of the mucous membrane, and the glandular structure becomes thereby very much thickened and hypertrophic, forming ridges and other excrescences on its free surface similar to that of the stomach. Or the secretory functions of the glands may remain, but as the secretions cannot ooze out from the cavities on account of the closed orifices by the enlarged and thickened epithelium, they dilate to an excessive degree and are converted into mucous cysts.

Epidemic Cholera.

The anatomical changes in epidemic cholera are characterized by acute inflammation extending over the whole intestinal canal, in which an enormous quantity of liquid is transuded. Within the intestinal cavity of those who die shortly after the choleraic attack there is found a very abundant opaque grayish-white, inodorous alkaline liquid, in which float very many opaque-white flakes or floeculi. The mucous membrane is injected, rather thick, moist, of a pink color, the serous tissue is also somewhat opaque. After death the epithelium exfoliates very shortly, the follicles are found more or less swollen, either grayish-white or of a very bright red color. Besides the epithelial exfoliation the microscope shows cellular infiltration into the mucous and sub-mucous tissues. The serous membrane is often similarly affected. When death takes place in the later stages of the disease, the intestines contain but a small quantity of a more condensed liquid, usually mixed with bile, sometimes a little black blood. In the colon hard fæces are often found. The mucous membrane is either pale, of a drab or a deep gray color, or green (sphacelus), and intensely injected, and perfectly impregnated with hæmorrhagic extravates. In the colon, in the lower portion of the ileum and in the sigmoid flexure many ulcers of different depths and extent may exist. In the colon alterations similar to those found in dysentery are very often found.

Abdominal Typhus.

Typhoid enteritis is an infectious disease, produced, according to *Klebs* and *Eberth*, by invasion of the typhoid bacillus. It is chiefly in the lower portion of the ileum and in the upper portions of the colon where the anatomical alterations in this fever are found. Very seldom are there any changes above or below these parts. It constitutes essentially an infiltration of the follicular apparatus and its surroundings, which soon become mortified, whilst it is accompanied by a catarrhal inflammation of the rest of the mucous membrane. In the first days of the course of the disease the mucous membrane in the lower part of the ileum, as well as its follicular plaques, are intensely red and uniformly swollen. A little later the inflammation increases in these plaques more than anywhere else, and there are formed raised pappillæ and ridges, which have some resemblance to the cerebral convolutions in their arrangements and configuration. The swelling extends sooner or later to the whole plaque, and numerous ridges are formed thereon.

When the swelling has reached its highest point then the whole surface becomes even, and only little depressions, being here and there, indicate the position of the follicles. On the surface, in the place of the solitary follicles circular nodules are formed. At this stage both the agminated and solitary apparatuses pass from an intense red into a grayish white color, which gives them the appearance of soft tallow, or cerebral matter. This swelling is produced by high-graded infiltration of lymphoid cells, both into the mucous and sub-mucous structure (medullary tumefaction of the older anatomists). The glands are displaced and separated in the mucous membrane by the excessive infiltration. The villi are equally swelled and infiltrated.

The sub-mucous is uniformly impregnated with the cellular extravasates beneath the whole plaque, so that they gradually lose their form altogether. Both the muscular and in very severe cases the serous coats become equally infiltrated. The number of the follicles and the extent of affection of each is very variable. A few are sometimes, or even only one inflamed plaque may be, the whole extent of the affection; or the whole intestine, till way up in the jejunum, or down into the lowest part of the rectum, may be thus involved.

In the second week of the course of the disease partial mortification of the swollen plaques takes place. (Tab. XIV, this Section.) Either the whole or parts of these are gradually destroyed. The surface of the plaques assumes a jagged appearance, and turns either yellow, grayish yellow, or dark brown, from the quantity of biliary pigment imbibed. Little by little the dead portions of tissue loosen from the still living and are detached both from the bottom and the sides. The surface thus denuded presents a smooth and fairly clean appearance. Its edges still remain for some time raised and swollen. As a rule the typhoid ulcers are confined to the solitary and agminated follicles; very seldom does the ulcerative and sphacelus process pass beyond the follicular apparatus. Occasionally there are some ulceration and destruction in the ileo-cecal valve and its surrounding mucous surfaces. In the follicular portion both the mucous and sub-mucous structures are, as a rule, involved in the ulcerative destruction. The muscular coat generally is preserved. But when it is strongly infiltrated then it becomes disintegrated as well as the other tissue. The serous tissues, as a rule, are the least involved of all. Even when it is much affected

PERFORATIONS, ULCERATIONS, GANGRENE OF THE INTESTINES, SOFTENING OF THE MUCOUS MEMBRANE.

Fig. 2.

Fig. 1.

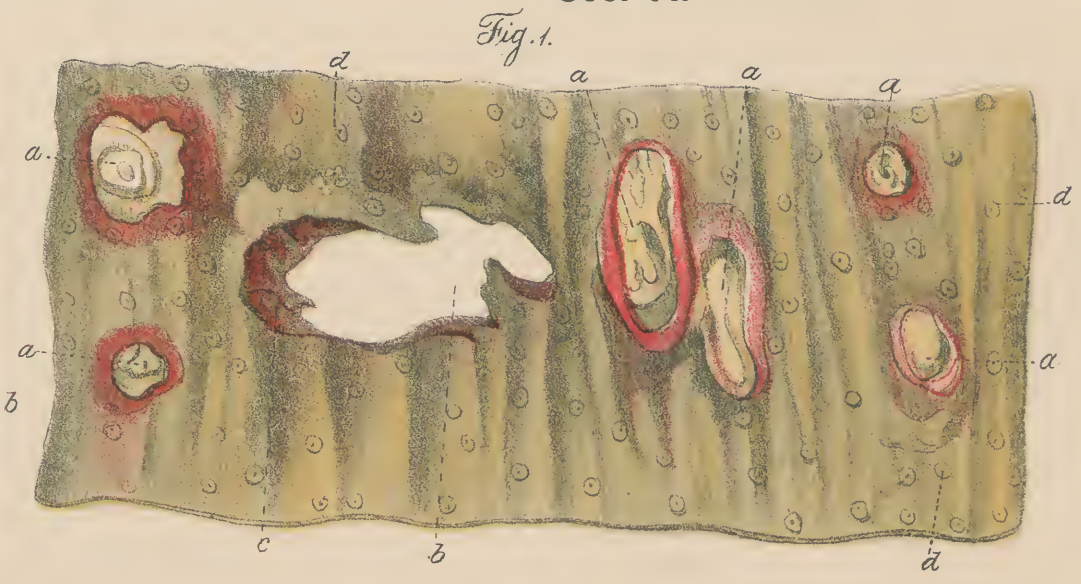


Fig. 3.

Fig. 4

Fig. 5.

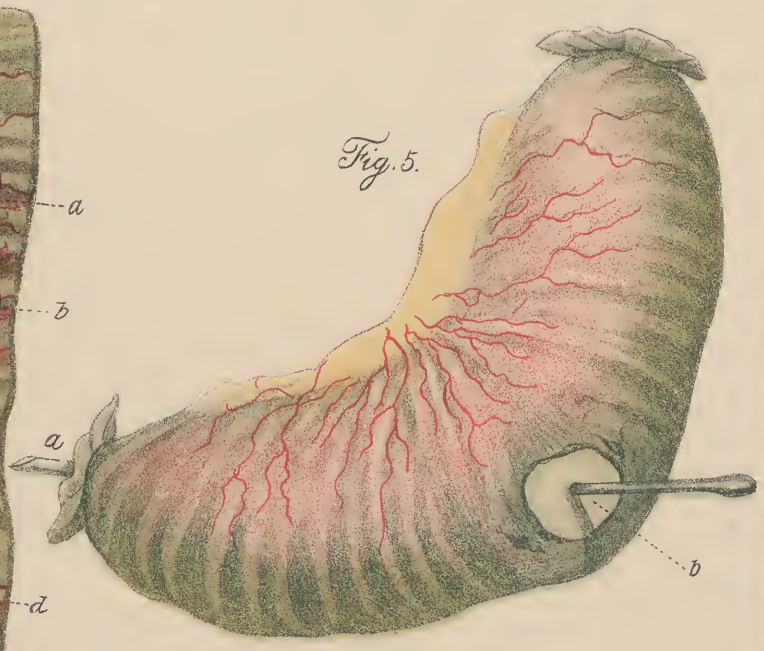


Fig. 6.

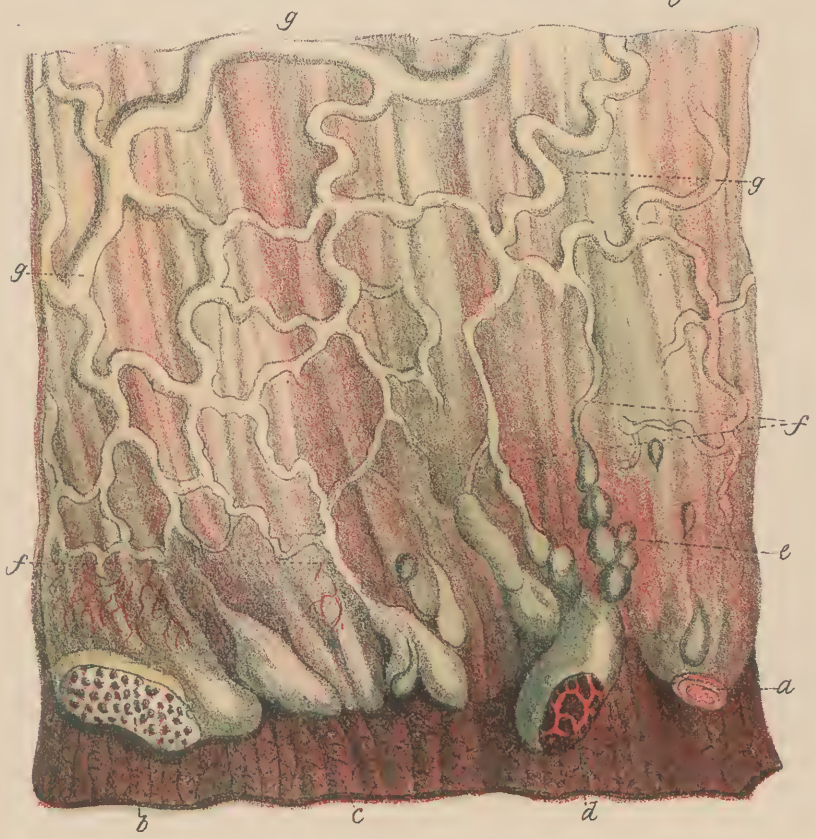
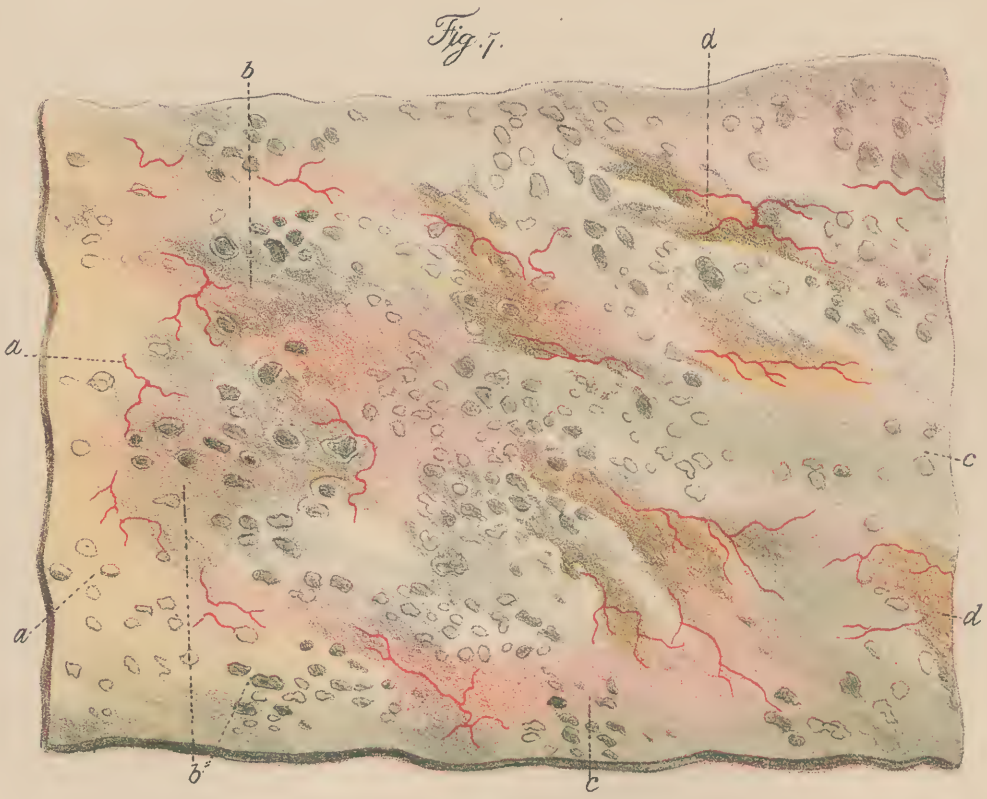


Fig. 7.



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Sec. VI. *Tab. XIII.*

EPIDEMIC DYSENTERY.
HAEMORRHAGIC FOLLICULAR ENTERITIS.

Fig. 1.

Fig. 2.

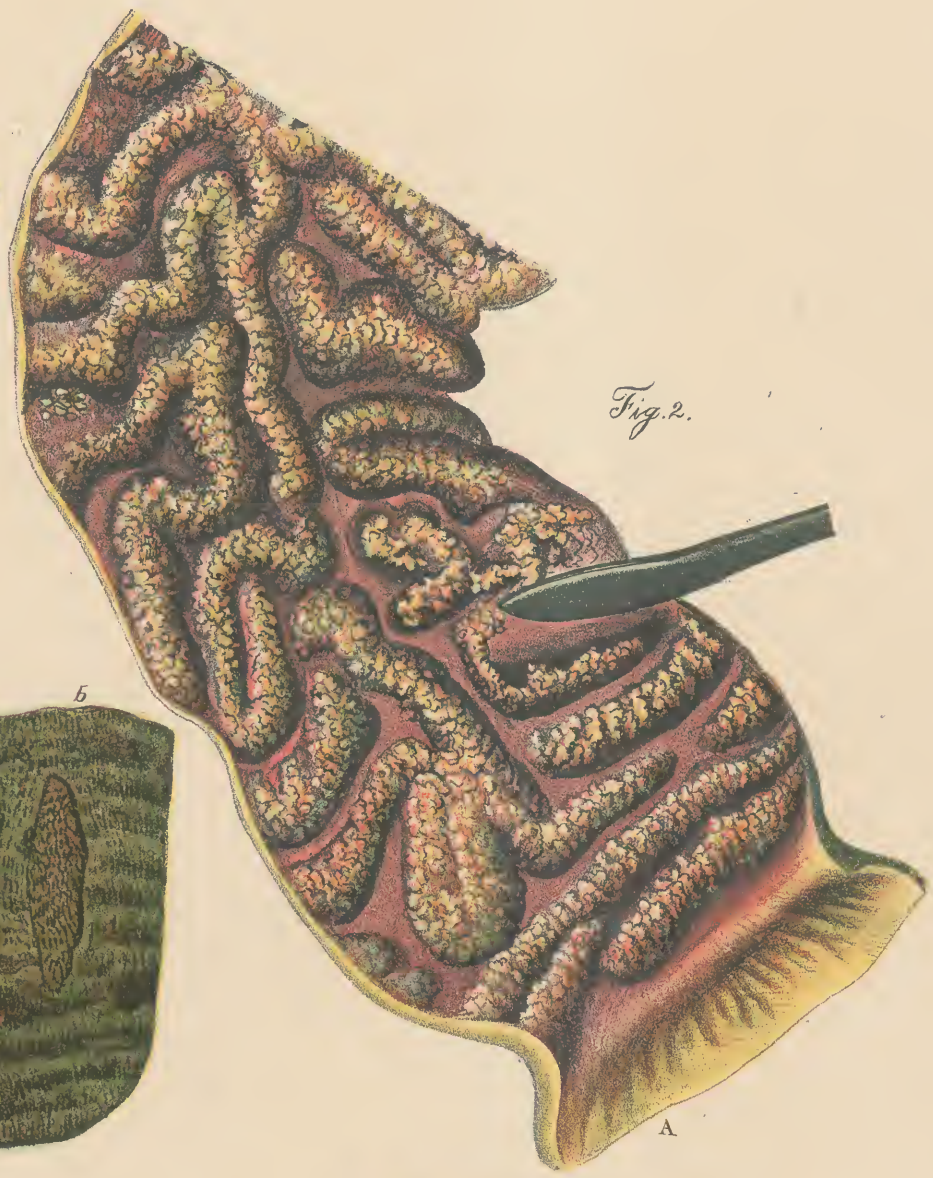

Fig. 8.

Fig. 3.

Fig. 4.

Fig. 5

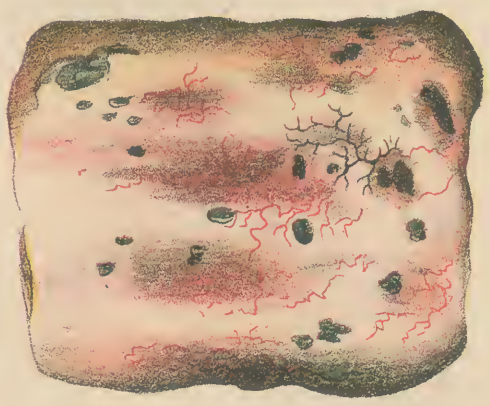

Fig. 6.

Fig. 7.

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TABLE XIII.

DYSENTERY, ENTERIC DIPHTHERIA. INTESTINAL GANGRENE.

FIG. 1.—*Acute Epidemic Dysentery.*

Case.—A man forty years old was taken with severe diarrhœa, offering the following symptoms: A high state of delirium, quickly relieved by an opiate. Some fever next day. Seems to gradually improve. Sudden development of very acute parotitis. Dies on the eighth day of the attack. Post mortem: Abdominal cavity. The liver is lifted up by an enormously-dilated gall-bladder, covering in front the ascending colon, its fundus reaching down to the coecum. The small intestines, the coecum, and ascending colon intensely inflamed. The mucous membrane is of a very deep red color, covered with a grayish-green exudate in small patches and flakes; and adherent to the membrane. The ileo-coecal valve seemed to form the focus of the intense inflammation; the follicles and the mesentery being but little involved.

FIG. 2.— The colon of an individual who died from an attack of very acute dysentery. The mucous membrane was intensely red, and formed many irregular folds, the crests of which were covered with white flaky exudate. The deep red color was due to extensive imbibition with coloring matter of the blood and bile, and to hæmorrhagic infiltration into the sub-mucous tissues. (A), anus.

FIG. 3.—The lower portion of the small intestine presents in the ileo-coecal region a state of corrugation of the tissues, forming very thick folds of the exceedingly infiltrated mucous membrane. They were covered with an exudate, variously colored by biliary coloring-matter. The ileo-coecal valve was exceedingly thick, and raised far above the surface of the membrane. The agminated glands were also very much swollen and raised above the surface; corroded in some places, and covered with some exudate. The same was the case with the solitary.

FIG. 4.—*Peracute Dysentery with Gangrene.* Patient affected with epidemic dysentery, with the following symptoms: Involuntary evacuations, blood mixed with much mucous, and very fœtid. Small, slow pulse. No hiccough. Died suddenly in a collapse. Post mortem: The whole of the large intestine exceedingly enlarged and thickened, the mucous membrane thrown in thick, irregular folds, the internal surface presenting a gangrenous appearance and forming a dark, thick layer, as if affected by a very corrosive mineral acid. The sub-mucous and muscular tissues, although very much infiltrated and very thick, not mortified. Neither the coecum nor the appendix as much injured as the other portions. The small intestine, the least of all injured. The rectum (R) presented the characteristic changes of the large bowels.

FIGS. 5, 6, 7, 8 show various alterations which the smaller intestines had undergone in the disease. Fig. 5, the least affected portion of the ileum, shows the mucous membrane to be smooth and even from the infiltrate; only the follicles are affected, and the vessels around them enlarged. Fig. 6, valvulæ conniventes of the duodenum covered with exudation, exfoliation of the epithelium, and denudation of the subjacent structure. Fig. 7, the epithelium exfoliated, the tissue below ulcerating, chiefly near the follicles; (AAA), ulcers; (B), the mucous structure, presenting a motley appearance. Fig. 8, a portion of the ileum, of which the serous tissue (A) is very thick, the sub-mucous, infiltrated and hæmorrhagic; (B), mucous tissue, covered with dark exudate; (A), Peyer's plaque in the middle of the figure, undergoing ulcerative destruction, epithelium exfoliated.

the mortifying destructions never reach the extent of that of the mucous membrane. When inflammatory and gangrenous disintegration pass upon the external membrane the result is nearly always fatal peritonitis.

The retrogressive, reformative processes may begin at any of the different stages of the disease. When no sphacelus is formed, or only to a limited extent, the infiltrate in the plaque is soon absorbed, the patches becomes soft and flabby, and static hyperæmia takes place in the soft tissues, which assumes a very red color from the imbibed blood serum and presence of extravasated red corpuscles. The whole plaque or solitary follicle, as the case may be, turns either reddish brown, grayish black, or deep red. The edges of the ulcers become thinner, softer, and equally hyperæmic. In such conditions very severe hæmorrhages and infiltration with blood into the tissues often place the patient in a dangerous condition.

When reorganization of the tissues commences, the ragged and flabby edges drop down upon the fundus of the ulcer, where a fine granular film is formed, which is soon covered with epithelium. Even long after the typhoid process has ceased, the ulcerated places present smooth, dark-colored depressions on the surface of the intestine. (Section VI, Table XIV, *all figures.*) Neither follicles nor glands are to be found in the dark remnants of the former plaques. Simultaneous with inflammations of the intestinal lymphatics there is an inflammatory process going on in the mesenteric glands. The color of the inflamed structures is, in the early stages of the disease, a very deep red; later, they assume a grayish or grayish-yellow appearance, from the enormous infiltration with lymphoid elements. (Section VI, Table XI, Figures b, c, d, e, f, g.) Still later, the infiltrate either becomes absorbed, when the inflammation ceases, or is transformed into a gray-colored sphacelus, which may, sometimes, also be absorbed, or become caseous, or calcareous. The spleen and the lymphatics of the neck are always more or less inflamed and infiltrated in conjunction with the intestinal lymph apparatus.

Intestinal tuberculosis is one of the most frequent affections of the tract, and is chiefly located in its lymphatic vessels and glands. (Section VI, Table XVI, Figure 1.) The colon and rectum are the next frequent localities for this lesion. The tubercle usually begins in solitary or agminated follicles, as a nodular elevation covered with epithelium. After a time there is formed in the center of a tuberculous agglomeration a yellow or yellowish-white speck (caseous change), from which the caseous and gangrenous destruction extends in the whole mass. Ulcers with thickened and infiltrated edges are formed, and spread more and more peripherally, by continuous spread of infiltration, followed by caseation, gangrenous destruction, and formation of confluent tuberculous abscesses. (Section VI, Table XII, Figures 4, a, b, c, d, g; and Table XVI, Figure 1, same set.) Large ulcers have a very irregular form, and are of various dimensions. Borders of such ulcers are usually infiltrated, but sometimes they are flat; or uneven nodular projections are found both in the fundus and in the edges, of a grayish-red or yellowish-red color, singly or in groups. The depth of such ulcers is variable. Not only the mucous, but also the sub-mucous coats are often involved when the tubercular process is very deep. (Sec. VI, Tab. XII.) Very seldom is the destructive process arrested, or does cicatrization take place. As a rule, the infiltration is continued in steady process until death puts an end to the destruction.

Syphilis of the Intestines.

Besides the broad condylomata on and around the anus there is often formed syphilitic ulceration in the rectum of females, occasionally extending from the anus upward, for many inches in extent. It is separated from the healthy tissue by a very sharp line of demarcation. The ulcer presents, generally, a very irregular surface, upon which there are some patches or islands of very boggy mucous tissue. Its edges are similarly undermined, for the inflammation is mainly going on in the sub-mucous tissue, and is the first destroyed. The ulcers freely secrete puriform liquid. The cause of the formation of such ulcers seems to be the infectious vaginal secretion. In the colon and the smaller intestines syphilitic ulcers are very rare.

Intestinal Tumors.

Hyperplastic formations, both malignant and non-malignant, are very frequent in the intestinal tract. Polypoid excrescences and vegetations are found of different sizes and forms. They are characterized by their containing a great quantity of glandular structure in their make-up, and generally constitute non-malignant glandular hyperplasia. In the small intestine they are less frequent than in the large, especially in the rectum. (Table XII, Figure 6.) By continuous peristaltic movement of the intestine, they are usually stretched, or the pedicle, if it is thin, will stretch very much, and protrude sometimes into the anus. Cystoid enlargements are frequently found in the body of the tumor. Carcinomata are the most frequent tumors in the canal, and chiefly in the rectum, the iliac, splenic and hepatic flexures, the colon, and the coecum. Rectal carcinoma often extends into the adjacent tissues, either downward, into the anus, or into the higher portions of the bowels. They are very rare in the ileum and jejunum. In the duodenum adjacent to or at the orifice of the ductus choledochus they are more frequent. They appear either as soft fungoid tumors or papillary excrescences of great extent. The intestinal wall usually suffers, even in the early stages of the lesion, extensive cancerous infiltration, whereby it becomes thick and dense. When the whole circumference of the wall becomes thus affected, the intestine turns into a rigid tube. The rectum is often found in this condition; the colon not so often. When the neoplasmata have undergone ulceration, their borders are covered with vegetations, after the decay of which the ulcers present an appearance not differing much from ordinary ulcers. Their edges and fundus may shrink by cicatrization, especially when the sloughing has involved the whole circumference. The adeno-carcinoma is the most frequent and the most malignant, for it involves all the tissues of the bowels. In small tumors, the excrescences still retain their tubular form, lined with cylindrical epithelium. But in further development they lose their original histological character, and form a solid mass of connective tissue, with only a thin epithelial covering. The colloid cancers are the next frequent malignant neoplasms, especially in the rectum. They form extensive vegetation upon its surface, with abundant cellular infiltration, into the intestinal walls. The least frequent is the schirrhus. Melanosarcoma is only found in the rectum. When carcinomatous ulceration extends into the outer layer of the intestines, hyperplastic inflammations are produced, causing adhesions (by formed vascular or non-vascular pseudo membranes) of the intestines and the inner abdominal walls. These adhesions often dislocate the intestines, and, not seldom, give rise to invaginations, etc. Cancerous ulcers are sometimes the cause of perforation of the bowels, with fatal peritonitis. Cancers also produce metastases in the lymph glands, the peritoneum, and the liver. Sarcomatous tumors are very rare, but lipomata and fibroid neoplasms are frequent. They may originate in any of the intestinal tunics, and often close up the cavity of the bowels or produce invagination and displacement.

Clinical Character of the Diseases of the Intestines, and Functional Disturbances in those Diseases.

As the several parts of the alimentary canal have different functions, of course the functional derangement will differ in its several localities. The duodenum being chiefly an organ of digestion, derangement of its functions will imply, mainly, disturbances of digestion. Morbid hyperæmia is characterized by hypersecretion of serum and mucus, which will, to a very great extent, prevent the full action of the digestive liquid. Duodenal catarrh is generally either associated with or consequent upon gastric hyperæmia or inflammation. The symptoms of disturbance of both of these organs resembling each other so much will often obscure the differential diagnosis. In either there is vomiting, pain in the gastric region, and often fever. Of course, if the pain be confined to the left side, the region of the duodenal flexure duodenitis may, with certain probability, be diagnosed. Icterus, even of a light degree, will still further confirm it; but still it must be considered that either temporary or permanent dilatation or displacement of the

TABLE XIV.

ENTERIC TYPHOID (TYPHOID FEVER).

FIG. 1.—*Inflammation of the Follicular Apparatus.*

Case.—M. N., a servant girl of seventeen years, at Guy's Hospital, London. She had been sick a week before entering the hospital, during which time she suffered mostly in her head. After admission she became unconscious and very delirious. Dark-colored stools. Is very feeble; subsultus of tendons. Died next day. Necroscopic examination soon after death showed the intestinal inflammation in the first stage; the aggregated follicles enlarged and surrounded by delicate vessels derived from the mesentery.

FIG. 2.—Lower portion of ileum, near the coecum, of a young woman who died from enteric typhoid on the eighteenth day of the fever, in the same hospital. The solitary follicles very much enlarged, and the adjacent mucous membrane very vascular. The plaques, transformed into solid brownish masses, are still covered with epithelium and stained with bile. (AA), portions of the plaques in primary stage of ulceration; the mesenteric glands exposed by dissection to show their enlargement.

FIG. 3.—Same intestine, slightly stretched and spread out in the light to show the dilated vessels running from the mesentery toward the follicles (CC, DD). The red color is due to the effect of light.

FIG. 4 shows the retroperitoneal surface of an altered plaque, showing characteristic distribution of the vessels.

FIG. 5.—*Advanced Stage of Enteric Typhoid.*

The case of a man of thirty-five years, who died on the seventeenth day of the fever. The whole intestinal surface inflamed; near the plaques (CC) are irregular ulcers, with raised borders, beginning to slough. The solitary glands (AA, BB) are differently affected; some are ulcerated, others in the form of pustules. The mesenteric glands (D) are much enlarged and very vascular.

FIG. 6.—Parts of an ileum of a woman of twenty. Was affected with a very high and protracted fever, the stomach and bowels in a constant state of irritation. Toward the last she was seized with severe pneumonia and erysipelas of the head. Died on the twenty-fourth day of the sickness. The intestine was gradually recovering from the primary effects of the fever. On its external surface there are two dark spots on the peritoneum (G H), corresponding to the ulcerated plaques on the inner surface. The membrane had not changed much, and retained much of its consistence and gloss. The mesenteric glands (I) are soft and contain pus.

FIG. 7.—Inner surface of the portion of ileum. The mucous membrane is very red and highly vascular. The ulcers (L K) correspond to the dark spots (G H, Fig. 6).

FIGS. 8, 9.—Enlarged figures of granulating ulcers and portions of the intestine exposed to the light. (AA, BB, C) show different portions, some granulating, some having dilated vessels, others formation of cicatricial tissue.

stomach may cause the pain, or even a light icterus, by dragging the ductus choledochus out of its place, and simply mechanically close the orifice, and for a time prevent the flow of bile into the duodenum. Dry and clay-colored stools of the patient in that condition will indicate disturbance of the duodenal function. Duodenal catarrh of the intenser kinds will produce not only the above-named symptoms, but very severe digestive derangements, especially lack of digestion of fatty and many amylacious foods, which are readily discovered in the eliminated fæces. Subjective symptoms, such as the aversion of the patient for fatty substances, great desire for acid or pungent foods, and pain in the duodenal region, are very unreliable.

Icterus, with gastro-duodenal catarrh, is usually manifested by the spread of the jaundice over the whole body, a small but not frequent pulse, often a feeling of great weakness in the region of the heart, usually numbness in the head, pallid, yellowish, either dry or but little moist, flabby tongue, and frequently great mental depression, muscular fatigue, and almost total anorexia. The gastric trouble is sometimes very prominent, nausea, retching, but little vomiting, sometimes much thirst, occasionally none at all; either obstinate constipation or very scanty, dry, clay-colored stools. Itching of the skin is a common phenomenon in this form of icterus. (The graver forms of icterus will be described among the diseases of the liver.) Normally the gall bladder is covered by the border of the liver, and no sound can be produced by percussion; but when any dullness of sound is produced in the region beyond the borders of the liver it constitutes a valuable symptom of closure of the orifice of the ductus choledochus and distention of the gall bladder with bile. Light forms of duodenal catharrh are transitory, and produce but temporary disturbances, but severer forms often end in ulceration, more or less permanent obstruction to the flow of bile, and often of the pancreatic juice, followed by general atony and marasmus, great loss of blood corpuscles, and poisoning of the nerve centra. The biliary components are readily discovered in the urine and often in the œdomatous effusion connected with disturbances of circulation in the portal system, which often are caused by intense gastro-duodenal catarrh with icterus. Causes of duodenal diseases are, as a rule, those of simultaneous diseases of the stomach, for idiopathic duodenitis is very rare. Gastro-duodenal icterus is ascribed by nearly all authors to closure of the orifice of the ductus choledochus, either by a mucous thrombus, or mechanically by a swollen fold of the intestinal wall. *Glax*, in a very able article, decidedly repels the idea of closure, but states that it is nearly always due to disturbance of hepatic circulation, for thereby an obstacle is placed in the way of the biliary ducts leading from the liver to eliminate the bile by way of the duodenum, and hence the freer passage of that liquid into the circulation by the central vein where it meets with no resistance. In many diseases of the liver this is certainly the case, but whether such is always the case in the duodenal catarrh or not seems to require further research.

Disturbance of duodenal digestion is liable to produce great disturbance in the alimentary canal, from the irritating action of the partly digested food, and especially fatty emboli in the smaller veins. Diarrhœa, or lodgment of the hardened fæces may follow with subsequent dysenteric discharges. In the spread of the catarrhal process into the common duct, the pancreas is very seldom affected. The subjects of these disturbances are, to a very great extent, uninvestigated as yet.

The disturbances of the intestines below the duodenum are such as befall all cavities lined with mucous membranes, and added to them are the derangements in their functions. Hyperæmia and hypersecretions, catarrhal, exudative and hyperplastic inflammations, are manifested by a variety of symptoms, which differ according to the extent and intensities of those disease processes.

In the smaller intestines hypersecretions invariably determine either partial or total suppression of absorption. Fluxes, alvine discharges of a watery, mucous, bloody or albuminoid, or sometimes puriform character are usually the prominent symptoms of those diseases. Fevers with typical or atypic oscillations of bodily temperature, nervous derangements, circulatory and respiratory disturbances, either from the nervous troubles or actual concomitant functional disturbances or anatomical lesions, are produced by the same cause as that of the disease of the intestine. Trophic and alimentary disturbances invariably exist even in the lighter grades of those diseases.

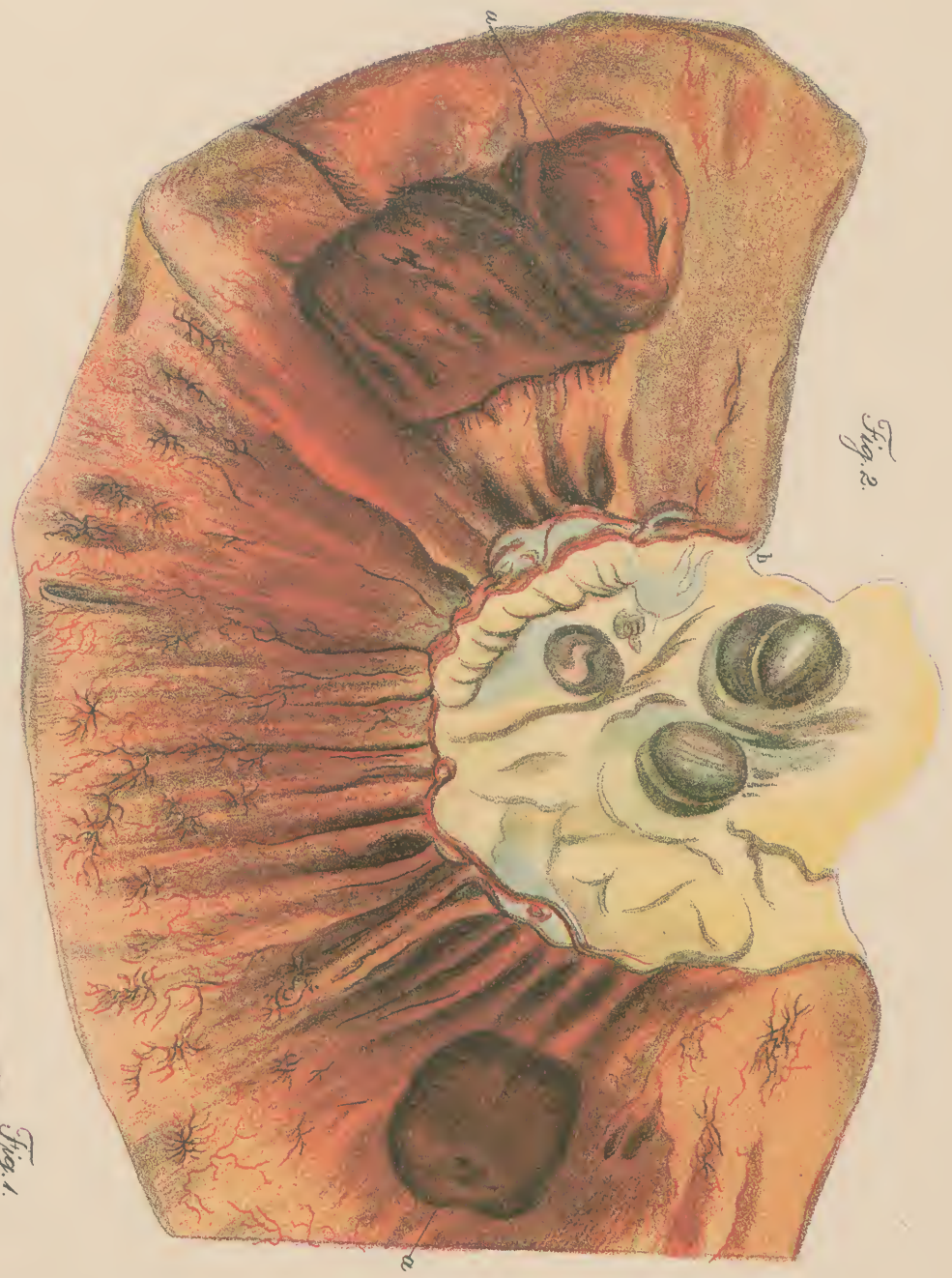
Diarrhœa. The substances discharged in diarrhœa are either those normally found in the intestinal cavities, or such as are derived from the blood, of which the serum transudes through the capillary walls, or such as either have been elaborated or exuded from the intestinal structures. Normally excrementitious matter consists of the undigested or indigestible substances, and the intestinal secretions, bile, and pancreatic juice. The greatest portion of the bile is reabsorbed in the intestine; the balance is changed in the process of digestion and eliminated (viz: dyslysine, choledoic acids, taurin, changed coloring matter). The cholates and choleates are only discharged when the food passes swiftly through the canal, in mucous diarrhœa or by the action of saline purgatives.

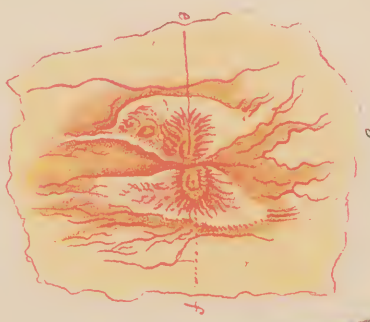
Fats and other substances which, acted on by several digestive liquids, are often found in considerable quantities; with these are cholesterine, fatty acids, acetic, lactic, butyric, a few traces of albuminates, and *excretin*. Inorganic soluble and insoluble salts, water holding those in solution or suspension in different quantities. (*Wehsarg.*) When the alvine fluxes contain only indigestible and unfit substances to be absorbed, there is but little or no harm done to the body, but when they carry with them important liquids or solids of the body there will arise a state of *inanition*, of atrophy and anæmia. Such are the consequences of the several forms of dysentery in adults and especially children. Copious biliary fluxes are usually harmless unless mixed with much mucus, etc. Mucous diarrhœa, *theoretically* thought to be a discharge of the greatly increased enteric juice, may be considered very problematical. An unhealthy organ secretes either very little or no digestive liquid; but there is often enormous secretion of mucus so-called, which, mingling with the copious serous transudate—usually taking place under such circumstances—is driven out of the intestine by rapid and painful peristalsis. Although there is pain, sometimes during or at other times shortly before evacuation, colic and tenesmus are not so frequent. This class of diarrhœa seldom assumes a very chronic form, except a permanent static hyperæmia supervenes by reason of vascular paralysis, such as obliteration or destruction of the coeliac ganglion. (*Budge.*) In its acute form, it is produced by either local vascular disturbance or general derangements of the circulation and nerve functions; under the influence of obstacles to circulation, from whatever cause; in diseases of the organs of respiration or circulation when the venæ cavæ cannot freely or sufficiently empty into the heart; in hepatic diseases, disturbing or obstructing the circulation in the portal vein. Intestinal fluxionary hyperæmia, consequent upon alteration of peripheral circulation, such as occur in extensive burns or scalds of the skin, call forth in the intestine at first simple catarrh of the mucous membrane and gradually a very dangerous and often fatal mucous diarrhœa. The most frequent cases of mucous diarrhœa are caused by the irritating action of lodged stercoral masses in the intestines. These, by remaining too long in the canal, undergo abnormal decomposition and fermentation by the living and chemical ferments always present in the whole tract. Great quantities of gases dilate the intestines, which itself hinders peristalsis and expulsion of the decomposing mass. Hypersecretion is invariably produced, which, with the dilated vessels transuding considerable serum and irritation of some of the sensory fibres in muscular tunic, produce sudden spasmodic movements of the bowels and expulsion of the whole offending mass. Local peritonitis is very frequently produced in such constipations (*Virchow*), which are usually followed by displacements, torsions, flexions, or stricture of the intestines, and thus inveterate the evil. Sometimes the intestines become enormously dilated, the abdomen tense, the diaphragm driven upward into the thorax, respiration made difficult, the arterial vessels compressed, congestions in the face and head. The patient having to continually dwell on his misery will become morose, discouraged, often hypochondriacal, and will intentionally, from fear of pain or diarrhœa, abstain from going to stool. Usually a brisk, reactive discharge improves all the troublesome phenomena. The stools are then either fœcal matter mixed with the liquid or even solid mucous strings or membranes enfolding the stercoral masses, or, in the intervals, glairy mucous, sometimes mixed with blood or some serum.

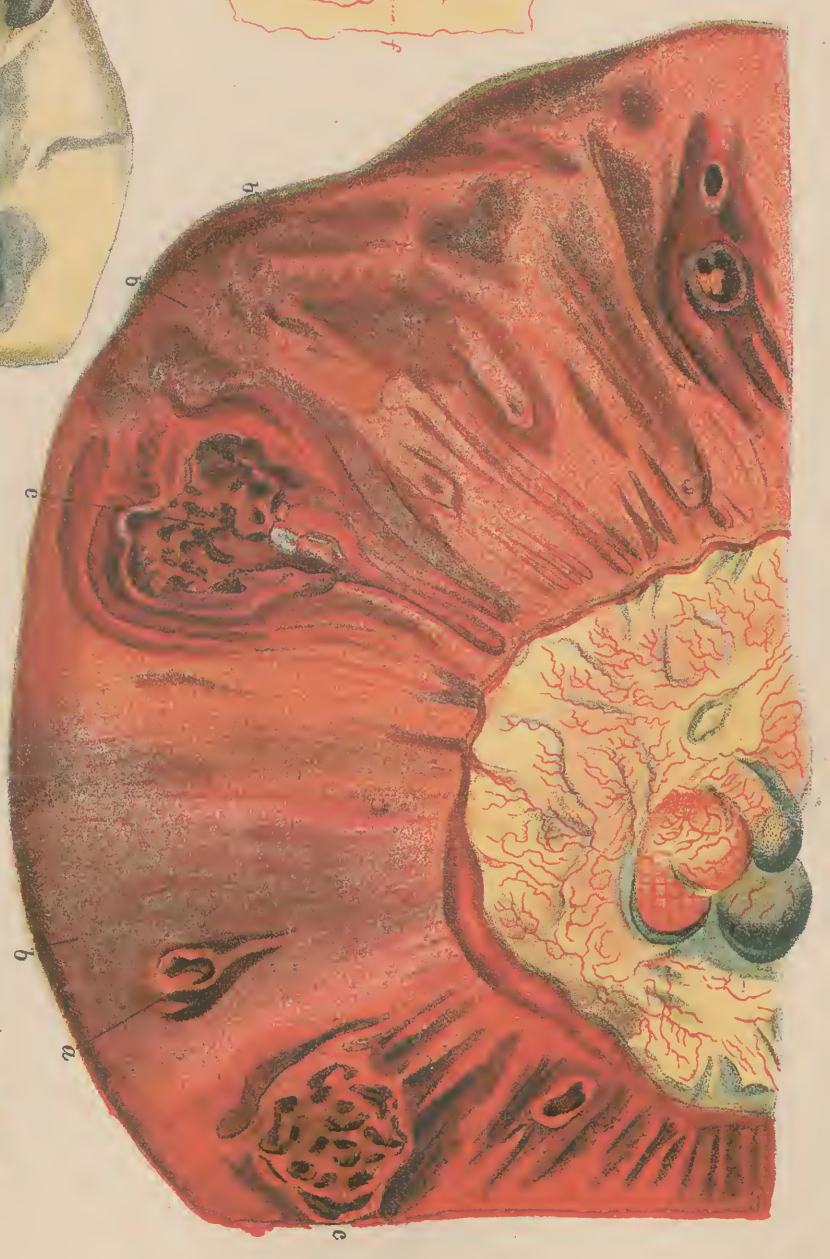
Serous Diarrhœa.

Intestinal transudates having the character of the serum of blood very readily take place. Saline purgatives, concentrated mineral substances, or in fact most of the ordinary cathartics usually produce such alvine discharges. They are composed of some albumen, considerable quantities of saline substances found in the blood serum, and great quantities of water. (*C. Schmidt.*) Pathologically they make up three different types of morbid discharges: the

ENTERIC TYPHOID.







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Sec. VI. *Tab. XV.*

EPIDEMIC CHOLERA.

PURPURA HAEMORRHAGICA.

C.

V. I. C.

A.C.

Fig. 1.

Fig. 2.

I.G.

Fig 5

E

Fig. 3.

F.P.

F

F.P.

A.C.

F

V. I. C.

F

f

f

C

Fig. 6.

Fig. 4.

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TABLE XV.

ASIATIC CHOLERA—HÆMORRHAGIC ENTERITIS IN SMALL-POX—MELANOTIC INFILTRATION.

FIGS. 1, 2, 5, 6.—*Epidemic Cholera.*

A portion of the ileum, coecum, and vermiform appendix of an individual who died in the early stage of asphyctic cholera. The mucous membrane is in some places intensely red, in others a little less so. The veins and arteries are turgid with thick, dark blood, especially near the follicles, some of which are so infiltrated as to appear as dark-red spots raised above the surface of the membrane. Fig. 2 shows a portion of the ileum, higher up than in Fig. 1, enormously injected, of a greenish color, having assumed that tint by imbibition with *biliviridin* in the intestine. The vessels are enormously enlarged and thickened. Fig. 6 shows a portion of an exceedingly-enlarged and thickened ileum, the follieles of which are infiltrated and surrounded with turgid blood-vessels. The plaque is enlarged, its epithelium exfoliated (E), the membrane in thick folds and of an olive-green color from imbibed biliary coloring-matter Fig. 5, part of ileum, coecum, ileo-coecal valve, and appendix. The solitary follicles (F F) are enormously enlarged, and project far above the pink-colored surface of the mucous membrane, usually found in the early stages of epidemic cholera. The vast number of miliary hæmorrhagic points visible over the whole small intestine (I G) show the great number of affected points. The vermiform appendix (A C) is thin and shriveled, the coecum (C) pale and in flat folds. The plaques (P T) are enlarged and corrugated, and highly injected. (V I C), ileo-coecal valve.

FIG. 3.—A portion of an ileum of a person who died of hæmorrhagic variola. The intestine is dark-colored and thick; great numbers of hæmorrhagic crosions are found on its surface, marking the situation of the agminated and solitary follicles.

FIG. 4.—Part of an intestine of a case of very acute hæmorrhagic enteritis. The tissues are enormously bloated from the great quantity of infiltrated blood, which turned a dark purple by the action of sulphureted hydrogen in the decomposing contents of the intestine.

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choleraic, the dysenteric, and typhoidal. Of the three, cholera diarrhœa is the characteristic type, as it presents great analogy with diarrhœa of purgation.

Epidemic Cholera. It is imposible to give an extensive description of the clinical phenomena of this dread disease in so narrow a scope as these compendious pages afford. Only the chief characteristics can here be sketched. The intestinal derangements constitute the most prominent morbid manifestations. In light cases the derangement is not very great, and is really the only important morbid process. Graver cases usually begin with some diarrhœa followed by intense attacks in a few days. There is nothing peculiarly characteristic about the diarrhœa, besides its being generally very copious, about eight or ten stools a day, which in the beginning have a rather bilious character, unless the discharges are very frequent at once. Usually colic and straining do not exist, but rolling and rumbling in the bowels are heard in most cases. Generally there is a loss of appetite. With the loss of liquids from the body, intense desire for drink and great prostration soon follow. Some cramps in the calves of the legs and huskiness of voice indicate its transition into the graver forms. It is conceded on all sides that, at this primary stage, the disease yields most readily to rational treatment. Very many people are thus affected in epidemics of cholera, and from the disease having that comparatively light form it has not been heeded and therefore neglected. Still the persons affected with this primary form, misnamed CHOLERINE, are able to move about, and thus scatter the seeds of the devastating cholera scourge. The discharges of such persons carry the infectious matter, whatever may be their nature. With this diarrhœa there is usually vomiting of a greenish, bilious, thickish liquid, and eventually a simple watery ejection from the stomach. Some pressure in the gastric region painful on pressure. Thirst becomes excruciating, distressing weakness, the heart's action at first nearly unchanged, the pulse rather frequent. Cholerine is the true transitory stage to true cholera. In the supervening asphycto-enteric stage, the discharges from the bowels change from yellow, bilious to a more watery form. The more frequent the dejection the more it loses its fæculent character and odor. This is the so-called *rice-water* stool. It constitutes a very watery, grayish liquid of 1.006 to 1.013 sp. grav., holding in suspension many floculi. It has a decided alkaline reaction. Besides the great quantities of water there are some epithelial cells of the mucous membrane, a few lymphoid corpuscles, some granular detritus, urea, traces of salts of potash and phosphates, but much chloride of sodium and carbonate of ammonia. The traces of albumen become only turbid by boiling. The micro-organisms present in the stool resemble those found normally in the lower portion of the intestine, and in common diarrhœa. Vomiting is nearly always associated with the purging. The vomited liquids resemble those of the stools; finally they pass off involuntarily both through the mouth and the anus. In so-called *cholera sicca*, dry cholera, when the discharges are absent, the *rice water* is found in the whole intestine in great quantities, with the same characteristics. The quantity of liquids thus emptied amount sometimes to one-fifth of the weight of the body. It is of course self-evident that such quantities cannot come from the intestines alone. The water is undoubtedly derived directly from the blood, for speedily all available liquids of the body, even pre-existing dropsical effusions, are attracted by the blood and then discharged through the intestines. Very seldom are corpuscular portions of the blood met in the stools. When found they bear the character of decomposed solid blood elements, and have a very fœtid odor of gangrene. That the rice-water discharges are transudates and not secretions, as *Conheim* claims, *Virchow* has clearly set forth (in his *Arch., Vol. XC.*, 1882), for the epithelial desquamation takes place very extensively during life, and traces of them are found plentifully in a very broken-down form. The whole coating of villi and larger patches have been found by *Rheinhardt* and *Leubusher* (*Virch. Arch.*, 1849, *Vol. II*). The modus operandi of the transudation lies in the actual participation of the veins in the process of exudation, as *Samuel, in his late dissertation on Cholera*, 1883, states: "Healthy veins absorb without any pressure whatever; those altered by inflammation exude, the entire catarrh increases the exudation and checks absorption, the exfoliation and hyperæmia are readily seen after death by the pink color of the mucous membrane. (Sec. VI, Tab. XV.) The serous exudate soon changes into a hæmorrhagic as seen in the same table."

Pathologically cholera is one of the most intense forms of enteric catarrh caused by microparasites, *the cholera bacilli.* They may act chemically, they may affect the mucous membrane, or may directly invade the capillary walls and then produce morbid alterations. Without the definite knowledge of the peculiar thalophite its action is difficult to ascertain; everything speaks in favor of the process being a mycotic form of fermentation and infection of the blood-vessels, to be due to the peculiar products of the ferments, for in the intestinal canal there are more than enough ferments to produce all forms of fermentation. Next to the intestinal symptoms are those of circulation in importance. The quantity of blood of course becomes exceedingly reduced by enormous watery depletion. It loses much of its sodic chlorides and becomes richer porportionately than normal blood, in solid substances, especially urea and urates. It becomes thick and condensed, and unfit for circulation. With the diminished quantity of blood passing through the heart there is a reduction of blood pressure, an increase of friction, and hence a lesser velocity of the circulation in the capillaries. The disturbance of circulation is early observed. The heart's action grows weaker with the increase of the discharges, fainting, beating of the heart, gradual disappearance of the apex beat; the cardiac sounds become more and more feeble, the first sound is changed into a murmur, the second disappears. Pericardial friction indicates a dry pericardial surface. At first the frequency of the pulse increases to 100, 120, or to 140, the pulse wave is lower, the pulse is thin, thready, occasionally imperceptible in the extremities.

Magendie and *Diffenbach* opened several arteries in that stage and found that they were but little filled, collapsed; the blood did not jet but simply leaked slowly out. Cyanosis indicates the venous condition of the asphyctic blood. The blood from an opened vein flows no more than from an artery. Post mortem: The heart is contracted, the left ventricle empty, the right containing some dark coagula and lardaceous erassementum. To the enteric stage the asphyxia is soon added, the blood can not circulate any more, extensive portions of the blood vessels are empty, and the distribution of the blood in the body becomes very unequal. The brain and the spinal marrow are the last to lose their blood. In the respiratory organs the same gradual enfeeblement and loss of function take place. The inhaled and exhaled air are nearly the same, but little absorption of oxygen and as little diminution of carbonic acid is carried on, which also hasten the asphyxia. The husky voice becomes feebler, is only a whisper, and then quite extinct. Death follows asphyxia. The temperature differs in different portions of the body, according to the quantity of the blood. The skin becomes dark, dry, and wrinkled from loss of moisture, but is occasionally covered with sticky sweat. Sensation in this *algid* state is gradually lost, and no pain or reflex phenomena, which were at first very pronounced and severe, such as muscular cramps in the bowels and lower extremities, are manifested now. All secretions are stopped and every function in the body finally ceases. The change for the better, if it takes place after the algid state, is manifested by cessation of the watery discharges and the vomiting. Sometimes this takes place wonderfully quick and indicates renewed absorption of the effused liquids in the intestine. Reaction begins with re-established circulation, cyanosis disappears, the peculiar characteristic cholera-expression of the face is changed, urination, which had ceased, is re-established, although with presence of albumen in the urine. In six or eight days polyurea usually ends. The skin resumes its normal function and is covered with warm perspiration. Thus enters reaction sometimes very speedily. Return of the attack is the rarest occurrence. The reactive phenomena are sometimes very violent, the pulse becomes very full and bounding, the vessels turgid, the head and lungs congested, delirium and fever appear. This constitutes the so-called cholera-typhoid. As a rule this does not last very long. Convalescence soon follows. Sometimes, when the nephritic troubles produced during the algid state are not corrected, the disease will assume a very grave, especially uræmic character, as there is formed great destruction of the blood corpuscles which the kidneys cannot eliminate, and dangerous glomerulitis takes place, with diphtheritic inflammation, which usually lead to death. Of course the vast changes of the blood very often lead to many sequelæ, and many cholera patients recover but very slowly from this terrible disease.

Enteric Typhoid.

Typhoid fever is produced by a thoroughly well-defined infection, the bacillus typhosus. Both its parasitory nature and its mode of invasion are known and studied. Besides their discoverers, *Klebs* and *Eberth*, very many pathologists have since made great researches into the subject, nature of the disease and its causes. Caused by a specific infection it follows its own special typical course and presents itself as a special disease form. The peculiar form of its localization in the follicular apparatus of the intestine and its well-defined, morbid phenomena following each other in nearly regular succession in the enteric inflammation have given it the name it bears—*Enteric Typhoid.*

Like cholera, the same infection acts with greater or lesser intensity upon different persons, of course the quantity of the infectious elements being at the same time taken into consideration. The lighter forms of the disease, like the graver, have a definite,

TABLE XVI.

TUBERCULAR ULCERATION OF THE INTESTINES — TYPHLITIS AND PERITYPHLITIS — LYMPHADENITIS.

FIG. 1.—Tubercular ulceration of the intestine of a man thirty-five years of age, who died of pulmonary tuberculosis. Nearly every viscus of the body was involved. The intestinal follicles were enormously impregnated with caseous tucercles, many of which were ulcerated. The ileo-coecal region was the most altered. The mucous tissue was largely exfoliated, the sub-mucous altered and thickened and to an enormous extent ulcerated; the coecal folds were enlarged, and the appendix adherent to the intestine. But little trace was left of the internal structure of the intestine. (A A), tubercular ulcers. (B), partly destroyed mucous membrane. (C), ileo-coecal fold.

FIG. 3.— Case of a young man of twenty-five years, who was suddenly seized with fainting, shortly after eating. He became speedily delirious, in which condition he remained until death, which took place the next day. Shortly before death he passed blood, black stools, and highly-charged urine. Post mortem: The lower portion of the small intestine sunk deep in the pelvis, of a deep red throughout. Peritoneum very vascular. Within the smaller enteric cavity some bloody mucus; in the larger, normal contents. The morbid changes confined to the ileo-coecal region. The mucous membrane was raised, swollen in large patches, and in many parts perforated. The plaques were the most affected portion in the ileo-coecal region. The folds (V I C) were largely involved. Between the raised patches the solitary glands were raised in the form of pustules (P P) and fine granulations (F F). The mesenteric glands were enormously infiltrated.

FIG. 2.—*Gangrenous Ulceration of the Follicles.*—A young man of twenty-four years, who died after a very short illness, and exhibited well-marked symptoms of highly-acute enteritis. Post mortem: There was found follicular ulceration, confined to the ileo-coecal region. There were very many follicles in a gangrenous state; the follicles were almost completely destroyed (E E). All of the tissues, except the peritoneum, which was but slightly affected, were in a complete state of mortification. The ileo-coecal valve (V I C) and its adjoining portion of the ileum were sphacelus and involved the whole periphery of the intestine. The ragged portions were detached and hanging in shreds and patches upon the intestinal surface. They were in a state of total decomposition, and very fætid. The muscular coat was bare. The cavity of the vermiform appendix (A C O), the colon, and the coecum were spread over with irregular-shaped ulcers.

FIG. 4.—*Perityphlitis.* Ulceration of the mucous and sub-mucous structures of the coecal folds, and a large portion destroyed by mortification. Quite a quantity of cicatrized tissue in a state of contraction, giving the surface a very uneven appearance. The borders of the ulcers very irregular and raised above the denuded muscular coat, which is covered with a very fine film of connective tissue. The whole surface was perfectly unfit for any function, from the total absence of functional structures. (A C), vermiform appendix. (V I C), ileo-coecal fold. (C), coecum.

FIG. 5. *Extensive Infiltration into the Mesenteric Glands, Follicular Ulceration.*

Case.—R. A., a man of thirty years, was sick three weeks with most pronounced typhoid symptoms; toward the latter part of the disease well-marked enteric symptoms. Post mortem: Brain and membranes but very little affected. Thoracic viscera but slightly altered. In the abdomen a great number of enormously-enlarged mesenteric glands, situated in the ileo-coecal angle. The sub-peritonial vessels are exceedingly dilated. The glands are some white, some reddish and injected internally, some form pockets filled with thick pus (G L O). The coecum (C) and the end of the colon present upon their inner surface ulcerated follicles. The ileo-coecal valve (V I C) and adjacent tissue are covered with very thick irregular folds, and show advanced conditions of cicatrization (P C) where the surface has assumed a ruffled appearance (P P). (I G), small intestines. (A C), appendix.

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typical course, only they are often overlooked from the very fact of the symptoms not being very prominent, and sometimes mislead the patient and the medical attendant to the great detriment of the former, whose life is thus put in jeopardy. Griesinger was the first to call attention to the lighter forms of the disease. He pointed out that "*the short duration of the sickness, and not the moderate character of the symptoms of a protracted attack of typhoid fever, ought to be the standard by which to gauge the gravity of the disease.*"

The most prominent symptom of enteric typhoid is the presence of fever. From the time of its commencement dates the course of the disease. Either a shaking chill or a rigor marks its beginning. This important symptom is often overlooked in the graver and very grave forms of the disease. In the lighter forms *Jurgensen* found in eighty-seven cases one-half the number to have begun with distinctly marked chills, or often repeated sensations of chilliness. Seventy-four of the eighty-seven persons affected could distinctly designate the time of its beginning. "*A sudden attack characterizes the lighter form of enteric typhoid.*" (Jurgensen.) *Wunderlich* has recognized three distinct stages of bodily temperature, which he designated as the *initial period* or first stage, the height, and the descending or reconvalescent stage. The initial stage of well-developed typhoid is marked by a gradual increase of the bodily temperature during the first three or four days. From the morning to the next evening an increase of about one degree centigrade. From the evening to the next morning a decrease of a half degree cent.; in the lighter forms no such oscillation exists. The rise of temperature is here sudden, and usually already on the second day. In the second stage the temperature in the lighter forms varies exceedingly. In the greatest number of these cases the typical oscillation of the graver forms are observed, viz: A rise of one to one and a half degree cent. from the morning to the evening, the highest temperature in the evening, the lowest in the later morning hours. The long intervals of remission contrasts in the lighter against the severe shorter periods of remission. Antithermic remedies prolong the cooling periods and often cause irregularities in the typical rise and fall of temperature. A singular deviation from the normal is often met in a sudden rise and continuation of nearly the same height of temperature in the shape of a continuous fever for about a week or so, with a gradual decline. In such cases swelling of the spleen, roseolar eruption, diarrhœa, slight bronchial catarrh facilitate the diagnosis of the disease very much. In rare cases the mitigated form is marked by relatively low temperature; from the second to the eighth day only twice forty degrees cent., ordinarily thirty-eight degrees cent., or some fraction of a degree, higher. The third stage is characterized by sudden fluctuations of heat. Morning sub-normal, or even normal, in the evening forty degrees cent. in the higher grades of typhoid; in lighter forms a barely perceptible rise and fall, with gradual return to the normal. The significance of the sudden rise and fall in the last stages of the graver forms is the great anatomical lesions which require time to be repaired by reactive processes of the body. In the mitigated forms the slight anatomical alterations require no such reactive measures and no sudden increase of temperatures. That lighter forms may become, by imprudence, faulty diet, irrational treatment, improper hygienic measures, exceedingly serious and often fatal cases, *Jurgensen*, *Ziemsen*, and *Immermann* have positively proven.

The spleen is found to be swollen and enlarged. Even in the lightest cases in about ninety-two per cent. Both in the severest and in the lightest this *splenitis* may not be diagnosed, but cannot be necessarily considered as not existing. The distention of the intestines, and often of the abdomen, may sometimes displace the whole or a portion of the spleen, and, of course, no splenic sound will be elicited by percussion. Roseolar eruptions usually exist even in the lighter forms of this fever, but its appearance and continuance depend on the gravity of the disease. In forty-six per cent. the exanthema appeared on the tenth day of the sickness. In seventy-five per cent. from the eleventh to the seventeenth day. The intensity of the eruption goes hand in hand with the intensity of the disease. In light cases it appears on the second; fourth, or fifth day. As many patients perspire very much (in the lighter forms) there is often found miliaria alba; erythematous spots are also sometimes observable, which very soon disappear. Bronchial catarrh is, with few exceptions, not very severe. The greater or lesser development of the bronchial catarrh does not seem to depend very much on the intensity of the typhoid process. In light cases there are few symptoms of pulmonic derangements, although very grave lesions often exist in the severe forms, which, however, are plainly manifested in the latter stages of the disease.

The enteric symptoms greatly differ in different epidemics; in those of 1865 and 1868 only twenty-one per cent. offered well-marked enteric disturbances of any gravity. Only sixteen per cent. of diarrhœa. In lighter cases, lasting about ten days, only twenty-one per cent., in more severe ones about twelve per cent. only suffered from severe enteric trouble. This shows that intensity of the disease is not in exact ratio to the enteric phenomena. Pain in the coecal region is *always* produced by pressure. Abdominal pains (colics) are very frequent. Lack of symptoms of intestinal trouble must, under no circumstances, be considered as absence of trouble of the intestines. In fact, next to the bodily temperature, the intestinal lesion requires the utmost attention of the physician *in all* cases.

Typho-enteric diarrhœa presents a mixed character. The stools, after staying a short time in a vessel, separate into two parts, one containing soluble saline substances (chiefly chlorides, having much affinity with cholera stools and likely of the same derivation) and soluble albumenates, as in dysentery. The sediment, usually mixed with biliary matter, contains mucous and a great quantity of ammonic-phosphate of magnesia; the latter not peculiar to typhoid stools alone. Within the debris of the tissue elements, voided by the intestine, great quantities of typhoid bacilli in all stages of development are found. (*Letzerich.*) Albumen is always present in the urine; in severe cases of the disease large quantities are found.

Ætiology of the Intestinal Diseases.

The highly alterable nature of the contents of the intestinal canal, both its secretions and the substances purposely or accidentally introduced into it, while it is eminently fit for carrying on the normal functions of digestion — that is, the proper change of all classes of food and secretions into absorbable chyle — becomes under unfavorable circumstances a source of diseases of those organs and of danger to life. Normally the innumerable ferments, both corpuscular and chemical, ever present in the bowels, only favor physiological function by creating solvents, by assisting decomposition, and bringing forth new affinities in the substances undergoing digestion. As long as the integrity of the mucous membrane is maintained, as long as no adventitious agencies injuriously modify its nutrition, that long do these active agencies of decomposition and alteration not affect the organs. But when those tissues become affected by inflammatory, mechanical, or toxic injuries, either directly or through the blood or nerve influence, then do these so useful forces become most detrimental and dangerous to the existence of the affected person. They become morbific agencies, having a short activity in acute diseases, and protracted action in the chronic. There is a class of exceedingly minute organisms, — although cognate with the harmless ones constantly present in the intestine, — which, when introduced into the body, become a source of special morbid processes, with typical manifestations all their own, and which make their appearance in epidemics *by an*

DISEASES OF THE ORGANS OF DIGESTION.

TUBERCULAR ULCERATION OF THE INTESTINES.

TYPHLITIS.

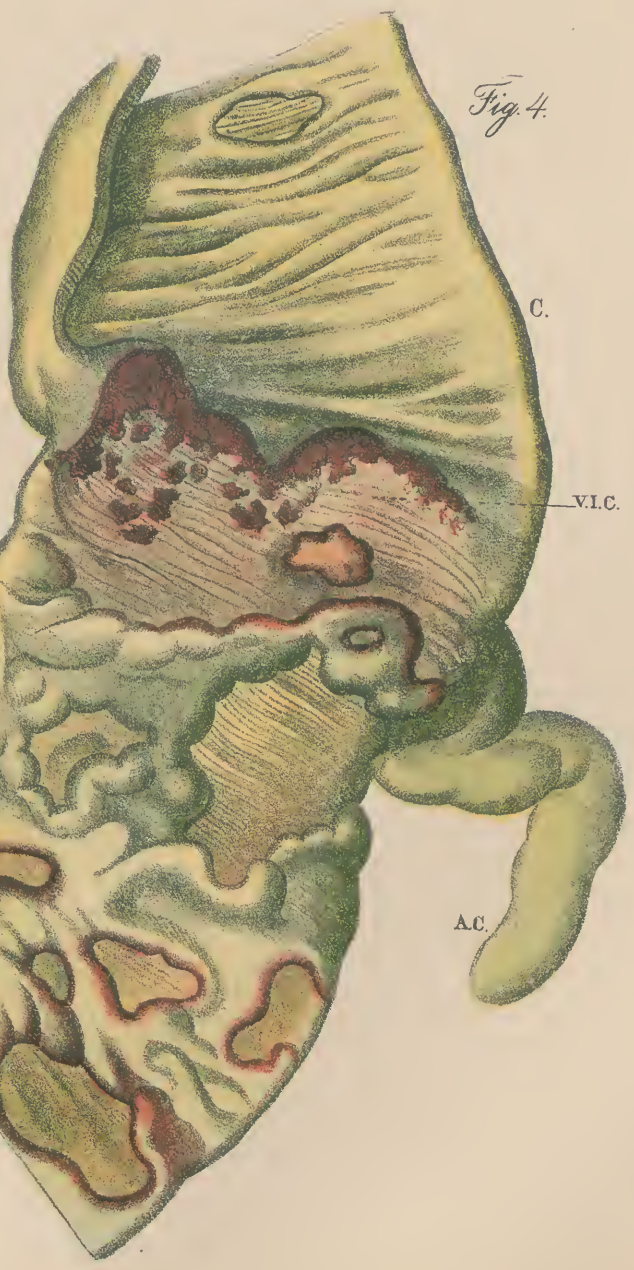
Fig. 1.

Fig 2.

Fig. 3.

Fig. 5.

Fig. 4.



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HEPATITIS. ATROPHIC CIRRHOSIS.

Sec. VII. *Tab. I.*

Fig. 1.

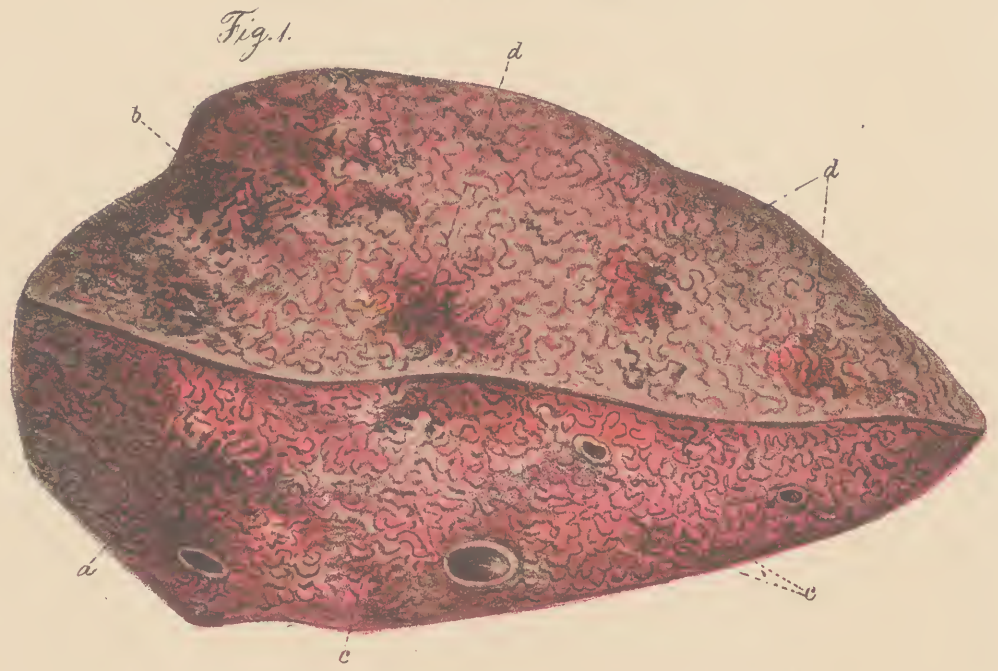

Fig. 2.

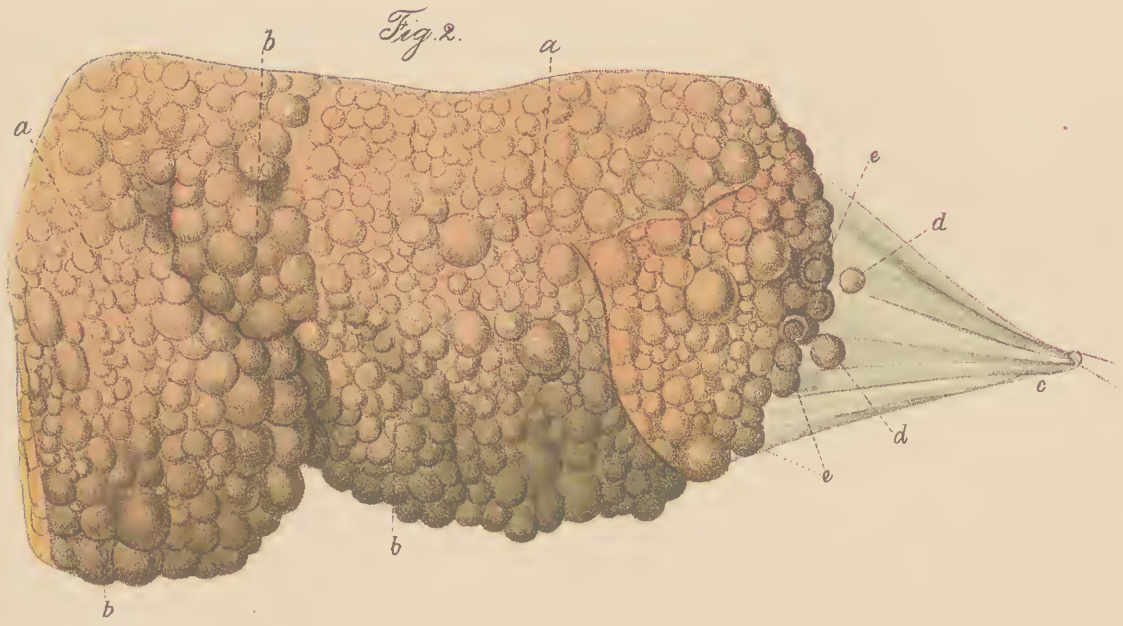

Fig. 4.

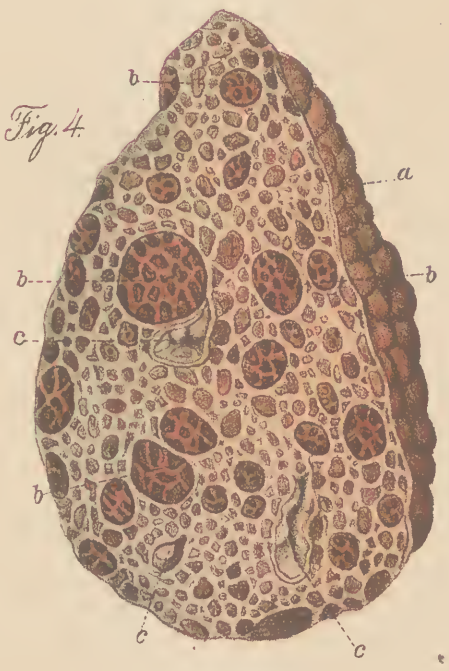

Fig. 3.

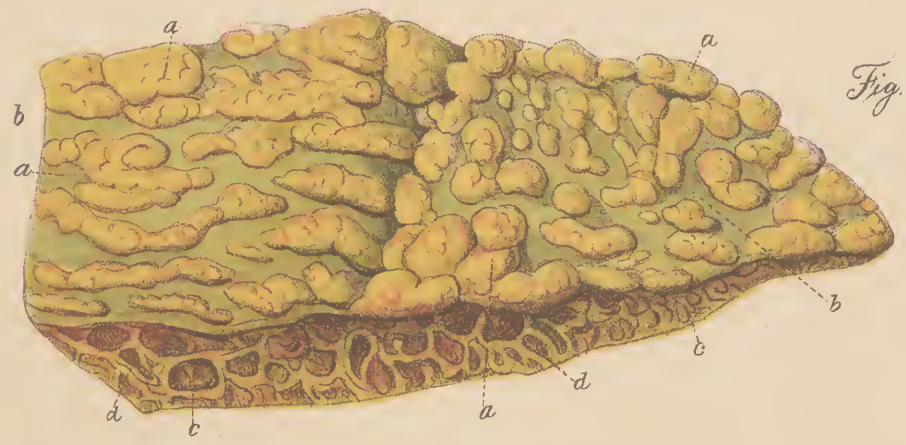

Fig. 6.

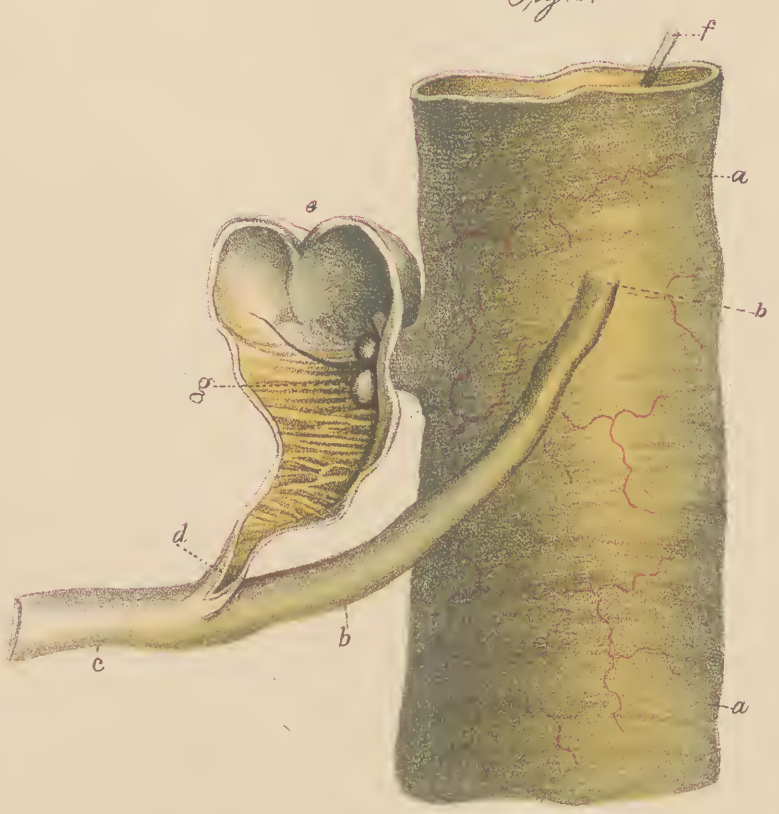

Fig. 5.

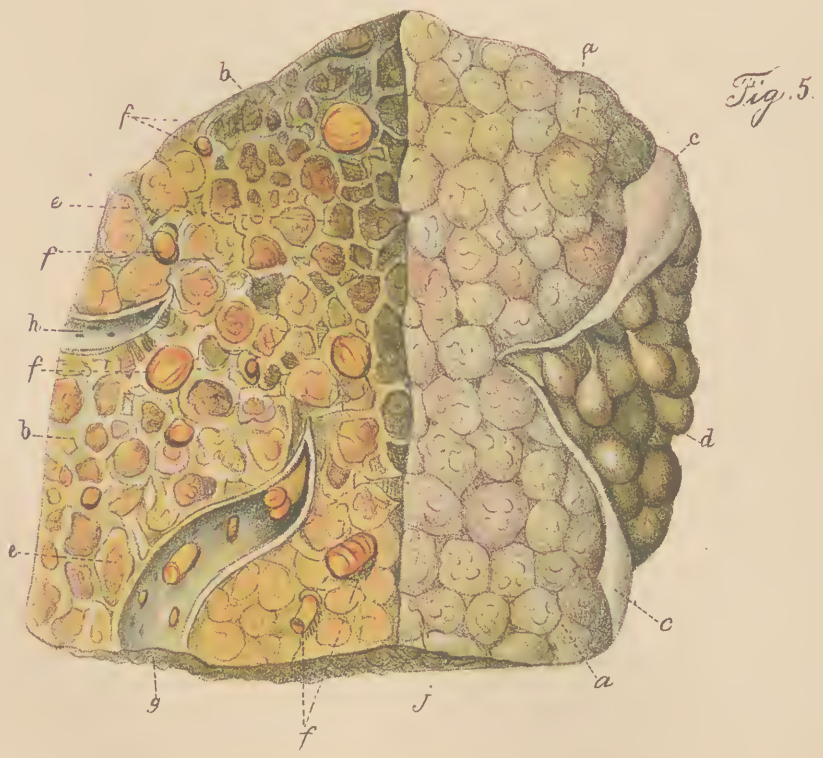

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TABLE I.

FIG. 1.—*Hyperæmia and Inflammation of the Liver.*

Besides diminution of consistence, which accompanies inflammation of this organ, the blood accumulates and is retained in the affected parts (congestive hyperæmia), thus obscuring the lobular structures. The redness produced by the hyperæmia becomes deeper and deeper as the inflammation advances until it is almost black. (Venous stasis.) These appearances are seen at (A, B, C C, and D D) the surface and in the substance of the liver. (*Carswell.*)

FIG. 2.—*Cirrhosis of the Liver.*

This figure represents the *atrophic* form of the disease, as produced by the presence of contractile fibrous tissue, formed in the capsule of *Glisson*, compressing the lobular structures. It shows the lesion in its early stage, when the quantity of the connective tissue is not yet very abundant, neither is the obstruction to the circulation of the blood in the organ nor effusion in the peritoneal cavity very great. (A A), tuberiform arrangement of the lobules seen through the peritoneal covering of the liver, of a yellow rust color; (B B), larger groups forming irregular projections; (C), a portion of the peritoneum removed to show the projections more distinctly; (D D), two of the round groups of lobules separated and suspended by the constricted vessels, the corresponding portions of the liver presenting two concave depressions; (E E), in which they were lodged.

FIG. 3.—*Further Advanced Stage of Cirrhosis.*

(A A), external surface of the liver studded with groups of lobules of an orange yellow color. The presence of fibrous tissue (B B) around these groups is very conspicuous from its great quantity and the light gray color; (C C), cut surface on which the openings of several veins (D D) are compressed.

FIG. 4.—A small portion of another liver affected with the same disease. The organ was much reduced in size; its external surface (A), of a yellow color, presented the tuberiform arrangement of the lobules, which is more conspicuous on its cut surface by the difference of the two classes of tissue. (B B B), groups of lobules of various sizes of a light or dark brown color. (C C C) show the compressed veins as described above.

FIG. 5.—A case of cirrhosis, in which the whole portal system of the liver was obstructed by coagulated blood, fibrine, and bile. (A A), external surface; (B B), cut surface; (C C), peritoneal covering separated to show the groups of lobules; (D), and their pediculated attachments; the same tuberiform arrangement of the lobules of the liver is well marked at (E E), where they are surrounded with neoplastic connective tissue; (F F), orifices of obstructed branches of the portal vein, by bright-colored fibrine stained yellow; (G), one of the veins laid open to show the obstructed orifices of its branches; (H), portal branch not obstructed.

In all the above cases there was ascites. In some of the severer and older cases there was anasarca, with general atrophy.

FIG. 6.—Atrophy of the gall-bladder from an abnormal communication between it and the duodenum. (A A), portion of the intestine; (B B), ductus communis choledochus; (C), hepatic duct; (D), cystic duct; (E), gall-bladder laid open; (F), small probe passed from the intestine into the gall-bladder through the communication, at the orifice of which are lodged two small gall-stones (G).

attack in force in whole regions of country. Cholera, enteric typhoid, dysentery, etc., are the names given to the collective activities exerted by them, upon the intestinal tract especially. Botanically they are classed as protophytes, and bear the somewhat indefinite generic names of schizo-myecetes (fissiparous fungi). In typhoid fever definite species of microphytes (*typhoid bacilli*) have been found time and again in several parts of the affected organs. In fact, their constant presence in the diseased portions, their appearance and disappearance corresponding to the progressive intensity and gradual cessation of the disease, and their constant localization in the follicles, connect them with the morbid processes going on in those organs. Although their mode of invasion has as yet not been definitely ascertained, yet their development in the ground-water in localities where there is a lack of drainage and in wells, cisterns, or springs, where liquid manure or other vegetable substances in a high state of decomposition exist, and the constant recurrence of attacks of enteric typhoid upon persons using such infected water, added to the fact that the same micro-organisms are found in the tissues, and that with the same organisms obtained by culture outside of the body enteric typhoid was artificially produced upon animals,—all these facts go to prove that the typhoid bacillus is intimately connected with the morbid process in that disease. The infection is produced by that *bacillus*. The substances, whatever they be, living or dead, if they contain this microphyte in forms capable of becoming developed when introduced into the animal body, and where they find conditions favorable to their development, will invariably produce enteric typhoid of one form or the other. The contagion, if such it may be called, exists only in this thalophyte. In any climate, any locality holding stagnant water of a nature favorable to the development of the *bacillus* in its several forms, can become a source of permanent affection. (*Klein, Socolof, Fischl, Rutter and Hubber, Walder, Eberth, Klebs, Letzerich, Wyss, Meyer, Koch, Litten, Almquist, Jurgensen.*)

The cholera infection is a transportable virus, and at home in the East Indies. It is very probably a microphyte; may be carried from there into all latitudes, and may for a time develop in any country, but nowhere is it able to permanently take position and become endemic or a centrum of propagation except in its place of origin, in *Lower Bengal* and in the bottoms and banks of the *Ganges* and *Brahmaputra*. (*Samuel, Die Behandlung der Cholera, Dissertation*, 1883.) Its mode of propagation is conveyance by ways of communications—by land and by water, in vessels, on railroads, in fact, by whatsoever mode the person or persons may travel and be transported when they are affected with cholera; and their dejecta, or substances containing or infected with dejecta holding the cholera-infecting agency, will become temporary sources of infection, and will spread according to the conditions favorable to the development of that noxious microphyte.

Dysentery.

Dysenteric diarrhœa presents a character and consequences quite opposite to serous forms of diarrhœa. The watery and the saline constituents are in very minute quantities, but in well-developed cases the evacuations contain a great quantity of albuminates mixed with much glairy mucus; in the farther advanced stages, fibrinous exudates, blood and pus corpuscles. Losses of such important constituents of the blood very well explain the great debility, the enormous exhaustion following severe cases of dysentery. Anæmia very soon becomes associated with a grave attack of even the acute form. Dropsy very often follows this disease.

Diseases of the Liver.—Anatomy.

The largest gland of the body, the liver, is an excretory as well as a secretory organ, and is supplied, for the performance of both functions, with circulation from two distinct sources: by the hepatic artery from the abdominal aorta and by the portal veins from the abdominal viscera. The hepatic vein connects the returning venous current, from the organ, with the inferior vena cava. The liver consists of a great number of lobules, or individual glandular organs, which are conjoined by a vast net-work of larger and smaller vessels, their perivascular structure and a very ample frame-work of connective tissue, in which the liver cells, the blood and lymph vessels, the biliary ducts, and the nerves are imbedded. The center or axis of each lobule is formed by a branch of the hepatic vein, from every part of which are derived great quantities of radiary branches, and these are connected by a vast net-work of anastomoting capillaries, in the meshes of which lie the liver cells in such a manner as to bring every little group of cells in immediate contact on all sides with the blood vessels. In the interlobular spaces and covering each lobule is a net-work of vessels, branches of the portal vein and their capillaries, which anastomose with those of the central vein, branches of the hepatic artery, with its abundant ramifications, and the larger and smaller bile ducts. The portal veins, which are the largest, the hepatic artery, and the bile ducts run parallel to each other in the interlobular spaces, mark the limits of the lobules which they surround and are enveloped in a quantity of connective tissue, derived from the perihepatic structure, the membraneous continuation of which constitutes the *perilobular* or *Glisson's* capsule. A section of the liver, perpendicular to the axis of the lobule, shows a spotted yellow and brownish appearance. The liver cells of the lobular center are more yellow than the brown venous zone. The lymphatics constitute fascicles, and with the branchlets from the nerves of the hepatic plexus are sheathed in a firm layer of connective tissue, coming from the hepatico-duodenal ligament. The liver cells produce glycogen; within the biliary ducts the biliary components are excreted. That the liver cells should at once produce glycogen and secrete bile is a physiological impossibility. It is perfectly true that any glandular cell may at times—when certain useless or noxious substances circulate within the blood, even if they were produced in the body itself (urea, urobilin, glycogen, glucose, many poisons, etc.), or when even normally useful substances accumulate in great quantities in the circulation—perform the work of elimination or excretion; yet that such double work should be a normal function seems very improbable. The liver cells certainly, like any other cells, and especially epithelial, are taking up great numbers of finely-divided particles from the blood, use them for certain physiological ends, and eventually eliminate them or remain impregnated with them for an indefinite period. The constituents of the disorganized blood corpuscles accumulate in the intestines enormously during the whole process of digestion, and are carried by the portal vein into the liver and a great portion of them are taken up by the glandular cells and elaborated into substances still useful to the body, or eliminated with the bile, which is most certainly constituted as a peculiar liquid within the fine biliary ducts in the manner of function carried on in tubular glands, and as *Henle* correctly states (in *Anatomy of the Viscera*, page 211), that "in the liver the finer biliary ducts are so covered with glandular organs, in the shape of appendicular tubules and racemous structures, that they appear more like channels carrying secretions from the glandular organs than anything else. If these little glandular organs were simple mucous glands, they would certainly exist in greater number in the larger ducts and in the gall bladder, where the mucus secreted would be needed for the protection of those walls against the action of the bile, which exists in great quantities in those cavities; besides their form greatly differs from those found in the depth of surfaces. Their enormous number certainly bespeaks for them a different secretion than simply mucous." Very many anatomical peculiarities found in morbid conditions of the liver are much in favor of this hypothesis. Besides the variable detritus of the blood structures the portal veins carry in the liver lobules great quantities of fat from the intestinal contents, where it is taken up in great quantities by cells and retained for a time in the periphery of the lobule, the vicinity of the portal circulation.

When large quantities of fat or fatty substances accumulate in the blood, or the fatty substances are not sufficiently used up in the body, there is always fat infiltration in the liver lobules. In well-fed individuals taking little exercise, nurslings, tuberculous and other consumptives of the lungs, in diseases of the heart with structural alterations, produced by venous stasis, in chronic alcoholism, the liver is always found impregnated with fat. In such conditions the portal zone of each lobule is exceedingly impregnated, the middle zone but little, and the central not at all, with fat. The difference of color in the several parts of the lobules has given this hepatic condition the name of *nutmeg liver*, which it very much resembles in its general appearance. More massive infiltration with fat nearly altogether

TABLE II.

FIG. 1.—*Red Atrophy of the Liver.* (*Virchow.*)

Case.—W. T., aged 66. *In Guy's Hospital, London.* History and symptoms: Was exposed in the open air, had lived rather hard, and became very poor. For the last four months his appetite had failed; about a month ago perceived that his legs had swelled and his abdomen became tumid. Had lost flesh. After admission the abdomen was distended and fluctuation very evident. The lower extremities anasarcous; countenance sallow; conjunctive slightly tinged with yellow; bowels open; pulse rather weak. Every part of the abdomen tender on pressure. Was completely broken down. Died 16 days after entering the hospital. Post mortem: Whole body slightly œdomatous. Abdomen swollen and fluctuating. No effusion in pleural cavity; slight effusion in pericardial cavity. Lungs partially but firmly adhered to the ribs and the diaphragm. Both lungs indurated, portions congested, some loaded with serum. Heart healthy, valves sound. A quantity of serum in abdominal cavity. The intestines atrophic but otherwise not much affected. The liver contracted and throughout of a morbid nature, apparently by deposition of minute portions of a yellow substance. The surface, covered by a very fine peritoneum, thinner than usual, presented a rough, granular, uneven surface, of a light-colored red and grayish yellow. A cut surface, of less red, but more yellowish color. The organ was rounder, thicker, but smaller than usual. The gall-bladder opaque and thick, containing a small quantity of bile. The common duct pervious but contracted at its entrance into the duodenum to an exceedingly small orifice. Spleen small and flaccid, light-colored, its capsule of cartilaginous density, small but natural in structure, but of a light color internally. (A B), portion of convex surface covered with peritoneum; (b), acute margin; (C), cut surface; (D), fundus of gall-bladder.

FIG. 2.—*Indurative Hypertrophy, with change of the Structures into Fat and Connective Tissue.*

(F), portion of convex surface of the right lobe near the acute margin; (g), cut surface.

FIG. 3.—*Amyloid and Fat Metamorphosis of the Right Lobe.*

In this case no dropsical effusion had taken place. The bile secreted was imperfect. In the two first cases (Figs. 1, 2) there was ascites; (h-i), portions of convex surface covered with peritoneum.

FIG. 4.—*Amyloid Degeneration of the whole Liver.*

Patient died with dropsical effusion preceded by icterus. The whole structure was changed into small rounded masses, varying somewhat in color. The secretion in the gall-bladder contained but little bile and was chiefly an albuminous fluid.

(K L), convex surface of the liver covered with peritoneum; (l m), cut surface; (K M), thickened acute margin.

FIGS. 5, 6. Both figures represent parts of the liver of H., a patient in Guy's Hospital, London. It was hardened and changed throughout its whole texture—an alteration having taken place in the parenchyma and in the secreting structures. Dropsy accompanied the diseased state of the organ. The power of secretion was somewhat preserved, so that the gall-bladder was moderately filled with yellow bile.

FIG. 5.—Part of right lobe covered with opaque adventitious membrane. (a), convex surface; (b), acute margin, thickened and rigid; (c), gall-bladder; (D), cut surface.

FIG. 6.—Part of same lobe, to show the internal structure.

The substance of the organ is here seen to be composed of two textures; the one cutting evenly, almost without any trace of peculiar structure; the other, in small rounded masses like enlarged or congregated *acini*. Both are intersected by bands of thickened cellular membrane. Some of the vessels are seen divided transversely. This was evidently a complicated lesion, of amyloid change with infiltration with lymphoid cells into the interlobular tissue, causing some compression of the lobular cells and infiltration and connective tissue formation in the capsule of *Glisson*, and extending upon the surface forming fibroid pseudo membranes. Infiltration with pigmentary substances and congestive hyperæmia is seen in Fig. 5, whilst in Fig. 6 infiltration with biliary pigment in the granular structure is plainly visible.

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obliterates the lobular boundaries, and the organ assumes a uniform yellowish-white appearance, and is rather soft. The volume of the organ is then increased whilst its spec. gravity is diminished. All its sharp borders become rounded, and the surfaces readily take the imprint of the ribs. The anterior border becomes very blunt and reaches the umbilicus. Such massive infiltration naturally leads to anæmia of the organ. Reabsorption of the fat by the veins or lymphatics can alone bring the impregnated tissue back to its normal condition, and that only by very powerful action of the heart. When this takes place the fat molecules coalesce and form rather dark brownish, large fat globules.

Amyloid degeneracy very much resembles the fatty infiltration as regards the locality in lobules. The substance with which the organ becomes impregnated is an albuminoid, and has, however, much similarity with vegetable starch in its chemical reaction. The earliest infiltration takes place in the hepatic artery region. The interlobular spaces at first remain, comparatively speaking, free. It begins in the smaller arteries and passes upon the cells which surround the capillaries. Gradually, as the disease advances, the region of the central vein is more and more involved, and lastly only does it affect the cells in the portal-vein region. The whole liver, in this condition, contains double its quantity of albuminates, which cause its increase in volume and specific gravity. The organ has a palish red color. Its tissue becomes transparent in the affected portions, and is of the consistence of beeswax. The quantity of blood in the organ depends on the quantity of the amyloid infiltrate. Of course the more the infiltration the less blood in the organ, and the less can the organic function be carried on; the quantity of bile is diminished, and of course nutrition is endangered. The capillaries may remain for a long while perfectly permeable to the blood. Combined fat and amyloid infiltration are frequent, although their morbid causes may not be the same.

True hypertrophy, that is, actual increase of its glandular normal tissue, can only exist in few partly-destroyed but rebuilt structures. Augmentation of many of its elements are products of compensatory hypertrophy when portions of the organ are destroyed (mechanically) and the individual lobules increase in size to balance the loss of the functional structure in the other parts of the organ. In leukæmia and diabetes mellitus, or in persons living in hot climates a long while —very likely living high and having but little exercise—a species of hypertrophy or rather a form of cellular infiltration takes place. In the portal-vein region the cells are swelled, and assume by treatment with iodine a red color, which would indicate the presence of a greater quantity of glycogen. The middle or arterial region is infiltrated with fat, the central zone nearly normal.

Atrophic condition of the liver is a very frequent disease of the organ; for every disturbance of the processes of digestion, if existing for any length of time, will at once diminish the quantity of blood in the portal vein; and when insufficient absorption be associated with disturbed digestion—which is generally the case—a diminished quantity of arterial blood in the arterial circulation will ensue, and atrophic condition of the liver will follow. Atrophy from local disturbances in the hepatic circulation is also common. The morbid alterations in the organ will correspond to the causes of the atrophy. Lack of alimentation from strictures of the alimentary passages and organs, when no other constitutional diseases, inflammations, or fever exist, will cause a state of general atrophy of the tissues of the liver. The organ may shrink to one-half its normal size (especially the lobules), the capsule becomes flabby and wrinkled, the cells reduced in size and impregnated with yellow and brown pigment granules. The other structures of the liver are not near as much involved. The preponderance of the latter over the almost obliterated cellular constituents gives the organ a denser consistence than usual, and may be mistaken for an indurated liver. *Virchow* designates one form of atrophy of the hepatic parenchyma, produced by permanent venous stasis in troubles of the heart and lungs, as *red atrophy*. This constant static congestion, which is always followed by venous dilatation, superinduces thickening of the perivascular structures and compression of the parenchymatous tissue by the great quantity of the blood in the veins. This over-quantity of blood gives the organ a red appearance, which, in some cases, becomes dark brown. The atrophy involves chiefly the region of the venous congestion—the central-vein portion—the lobular cells decay, and in the shriveled elements blood pigment, red, brown, yellow, becomes most prominent. Lymphastasis and dilatation of the lymphatics are here, as everywhere, associated with venous congestion. Of course near the larger venous trunks the destruction of the cells is greater than in such where the vessels are smaller, and upon the surface of the liver deep furrows will indicate the lines of disintegration, corresponding to the course of the dilated veins. The uneven surface often misleads into assuming that a granular state existed in the organ, especially as in this form of atrophy development of connective tissue often takes place in many parts where the liver cells were obliterated.

Yellow atrophy, a rare form of *softening* atrophy, is characterized by distinctive anatomical features of the morbid change as well as clearly defined clinical symptoms of pernicious icterus. It is an acute process, and the decrease of volume of the organ may take place in a very short period, to one-third of the normal. After death of the affected person the liver is found to have exceedingly shrunk in its thickness, very soft and flabby, and its capsule perfectly shriveled. (*Zenker.*) Section shows a mottled appearance of red and yellow, the red portions surrounding the yellow, which form islands. The yellow portions are of a very bright tinge, very soft and spongy, and raised above the red portions, which are firmer, sometimes very dense and tough. The yellow substance forms the primary alteration, the red secondary or later stage. In the early stages the lobular structure is still plainly recognizable in the absolutely anæmic and highly icteric yellow tissue. The large venous trunks in the lobules are then the only blood vessels filled with blood, the interlobular small vessels are utterly bloodless, and mostly obliterated and replaced by interlobular connective tissue. Very many glandular cells are fatty degenerated. When this degeneration becomes general the characteristic lobular structures disappear more and more, and the whole lobular cell structure turns into a fatty mass, unequal in the different portions of the lobule in the extent of alteration. Both fat molecules and blood detritus—hæmoglobin, hæmatoidin, bilirubin—are diffused in a fibrous structure in which but few cellular elements are to be found. More and more are the cellular elements wiped out by fat—metamorphosis —and only remnants of the parenchyma are noticeable near larger vascular branches, and even these are full of pigmentary granules. Eventually, when the person thus affected lives long enough for a partial regeneration of the affected liver tissue, strings of connective tissue and numerous rows of peculiar cells make their appearance; the fat accumulation slowly disappears, massive invasion of lymphoid cells and pigmentary matter now takes place, and highly protoplastic, finely granular cells with many processes are formed. They are very likely the newly-forming parenchymatous cells. From extensive destruction in such an organ as the liver no one ever recovers, but when this form of lesion exists only in a small part of the liver there is now and then a partial regeneration of the destroyed tissue and formation of cicatricial structure, wholly unfit for function.

Circumscribed atrophies of the liver from mechanical injuries and local pressure, exerted upon parts of the organ by cloths, stays, girdles, belts, or displacements and enlargements of neighboring tissues, etc., are very common. These atrophic forms are usually found upon parts of the organ and noticeable by the change of shape and surface of the

SIMPLE ATROPHY, AMYLOID CHANGE.

INFILTRATION WITH FAT.

Fig. 1.

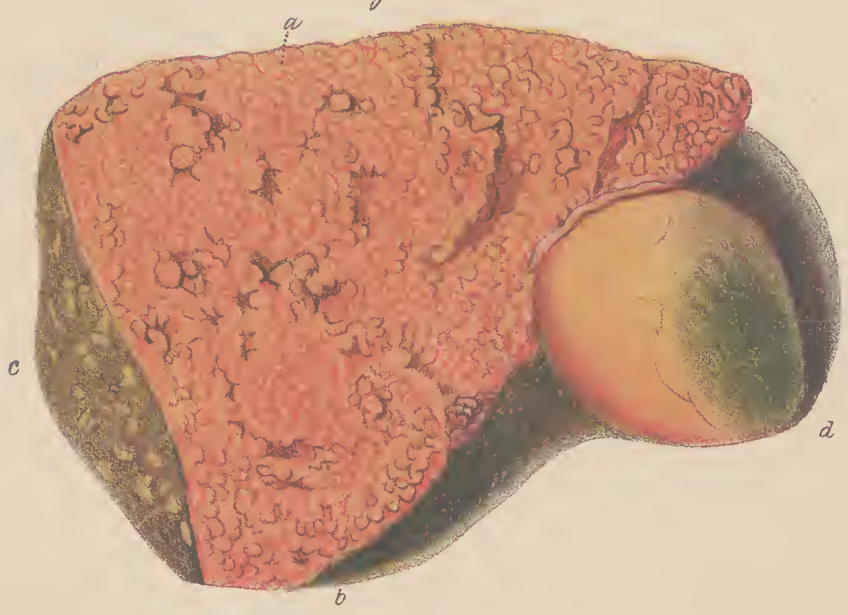


Fig 2

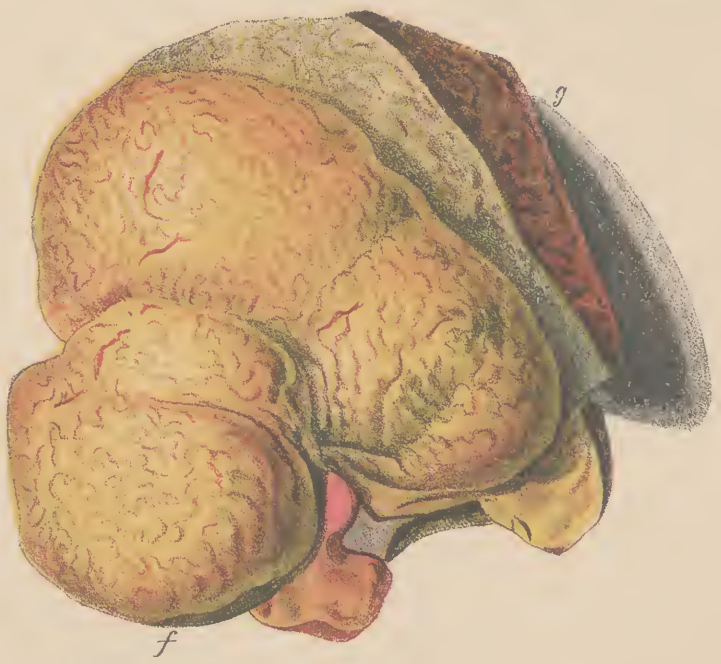


Fig. 3.

Fig. 4

Fig 5

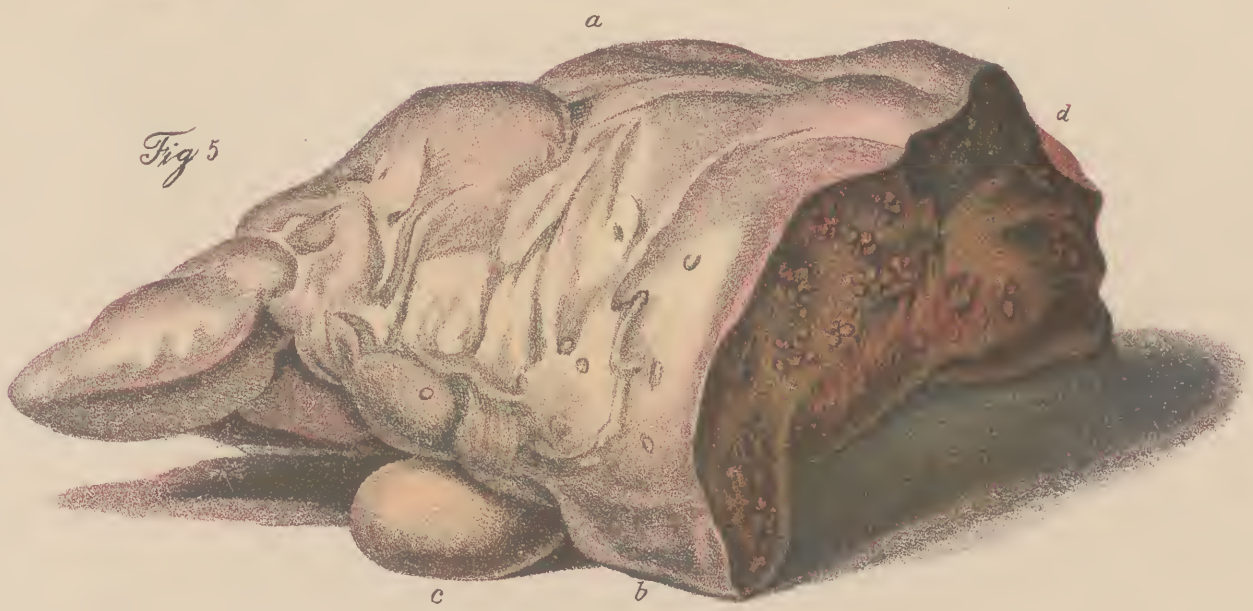


Fig 6

The Donaldson Lith. Co. Cincinnati.

Sec. VII. *Tab. III.*

Fig. 1.

Fig 2

Fig. 3

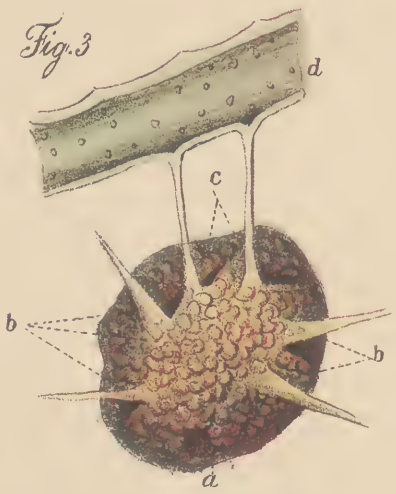


Fig 5

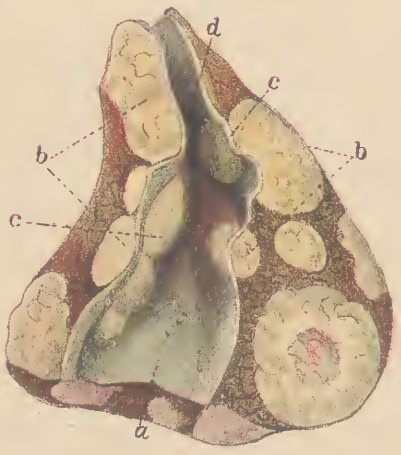
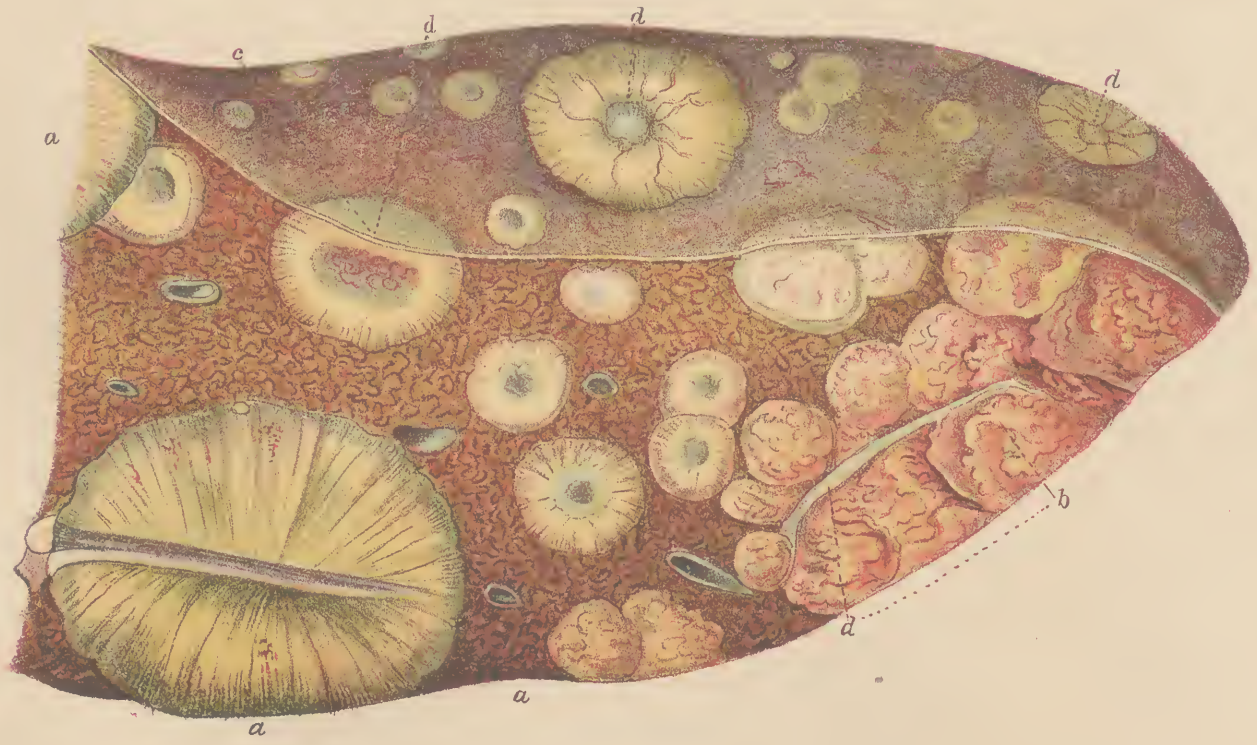


Fig 4

Fig. 6.



Thomason Lith. Co. Cincinnati.

TABLE III.

FIG. 1.—*Melanosis of the Liver.* (*Carswell.*)

(A A), Section of a number of globular tumors, varying from an eighth of an inch to an inch and a half in diameter, of a deep brown or black color, homogeneous aspect, uniform texture, of soft consistence, and lying in immediate contact with the substance of the liver. (B). Similar tumors but smaller in size. (C C, D) represent appearances which sometimes are seen to precede the formation of such tumors. At (D) the dark-brown substance is contained in the minute veins, and presents, when closely examined, a ramiform distribution. The appearance shown at (D) also illustrates the fact of melanotic matter being contained in the blood of the venous capillaries prior to its deposition in the molecular structure of the liver. (The dark substance is reduced hæmoglobin and bilirubin.) (Ref.)

FIG. 2.—*Carcinomatous Melanosis.*

The carcinomatous alteration is recognized by its pale and yellowish-white color, the melanotic by its dark-brown tinge. Both give the structures of the liver a tuberiform arrangement. (A A), melanotic portions; (B), carcinomatous, degenerated tissue.

The organic structures are still conspicuous from the form and arrangement of the *acini*, but the color of these bodies has disappeared and their volume increased by neoplastic deposits. At (C) a quantity of carcinomatous structure is accumulated, and seems to be increasing in bulk by the aggregation of the neighboring *acini*, which form an irregular border along the inner side of the principal mass. The melanotic substances seem to be principally deposited in the interacinous portion of the organ, readily noticed at (D D).

FIGS. 3, 4, 5, 6 represent different forms of carcinomatous alterations in the liver. Fig. 3, a tumor (A) removed from within the liver substance, with a number of small veins (B B) adhering to it. These vessels passed through the tumor, and contained the same brain-like substance as the tumor. Two of the vessels (C C) communicated with a large venous trunk (D) also thus affected. Fig. 4, carcinomatous matter in a larger venous trunk, from a tumor (B), being a large branch of the portal vein (H); at (C) the vein passes from the tumor and receives a superficial branch (D). Into this branch three smaller branches of veins (E) terminate (F). The whole venous trunk is divided to show its carcinomatous contents (G) and its communication with the chief division of the portal vein (H). Fig. 5 shows the compressed state of a vein produced by carcinomatous infiltration of its own walls and perivascular structures, causing venous stasis and subsequent dropsy in the abdomen, etc. (A), a large-sized branch of the portal vein laid open; (B B), tumors projecting into it (C C) without perforating the vessel; (D), partly obliterated cavity of the vein. Fig. 6, lardaceous tumors of different sizes in the liver (A A). They are of a grayish yellow color, with a central depression from which fiber-like darker structures project in a radiary manner. The tumors contain quite a quantity of brain-like carcinomatous infiltrate. Minute vessels ramify in them, from the circumference to the center. (A B), are nearly all of the medullary cancer form, some containing very many miliary hæmorrhages; (A C), the vascularity is very conspicuous; (D D), tumors on the surface of the organ, visible through the peritoneal covering.

organ. In partial atrophy recovery usually takes place when the inciting cause is removed, for the blood vessels are under such circumstances but little affected, and the destroyed cells are readily replaced, when free circulation is re-established.

Even under normal circumstances does the liver contain about 11 per cent. of fat in the fresh state, and about 32 per cent. extracted with ether. After every meal there is an increase of fat in the organ, varying in quantity according to more or less fat in food and drink taken. Great quantities of fat accumulate in the organ in many infectious diseases and in high-graded fevers. The greatest quantity is found in livers of patients down with puerperal fever, poisoning with alcohol or phosphorous. Anæmia from compression of the vessels by the fat only takes place in *high-graded* fat livers; ordinarily anæmia in livers is due to other causes. Accumulation of fat in the organ is either due to infiltration or fatty metamorphosis: 1. When the destruction of fat formed in the liver from albuminates, normally taking place, is prevented by introduction into the organ of great quantities of carbo-hydrates (sugar, amyloids, etc.) 2. When fat formed in the liver does not become oxidized (as in typhus, inanition, pulmonary tuberculosis.) 3. When enormous quantities of fat formation take place in the liver, but inadequately destroyed (as in poisoning with phosphorous, acute yellow atrophy, etc.)

Many causes often combine to produce accumulation of fat in the liver. It is often impossible to determine between accumulation of fat by infiltration and fat metamorphosis.

Inflammation of the Liver.

Parenchymatous inflammation.—When the liver is moderately enlarged in all its dimensions, and is of a yellowish-gray color, anæmic, and of an inelastic doughy consistence, smooth on the surface, and dry on a cut surface, like smoked meat, it can be set down that there was morbid alteration of the liver cells, which *Virchow* designated as *turbid swelling*, and forms the essential basis of the parenchymatous inflammation. This morbid process consists of enlargement of the cells by very numerous protoplastic granules. Sometimes these granules are in enormous quantities, and hide the nuclei of the cells of the liver. The lobules are then enlarged, and the interlobular connective tissue infiltrated with serum. Between the lobular cells spaces are formed and their otherwise regular rows disarranged. The serous infiltration likely extends between the cell-rows. Diffuse parenchymatous inflammation, of a light grade, is found in many infectious diseases, in many acute eruptive diseases, in septic and other poisonings, puerperal fever, etc. As a rule, this form of inflammation terminates in recovery.

Purulent Inflammation. Abscess in the Liver.

Such inflammation takes place when the morbific agency, either introduced from without or derived from any part of the body, is capable of producing pus. Modern research has proven that only schizomycetes, and the products of their activities, are able to form pus. The ways by which such microparasites penetrate into the liver may differ, yet are easily conceived. Any perforating abdominal wound may afford them entrance, or an adjacent organ may be in a suppurative state of inflammation, and the infection may be derived from those, either direct or by the lymph-current.

Parasitory infections are oftenest carried into the liver by the portal vein and hepatic artery, very seldom by the hepatic vein. In new-born babes they are conveyed by the umbilical veins. Especially is this the case when in the region of distribution of the respective vessels—the portal vein in the intestine, the hepatic; in the lungs; brain—there exists already a suppurating focus. Idiopathic abscess of the liver is very rare in temperate zones, but frequent in hot climates. The biliary ducts are often the ways of communication of the infection with the liver, when there is stagnation of bile in the ducts and formation of biliary concrements. When the *micrococcus septicus*, for instance, enters into the liver vast colonies are soon developed in the capillaries and in the veins. The zoogloea and the colonies soon obstruct and eventually close the vascular cavities. At first the parenchyma is unaffected, but finally the lobular cells become infiltrated with them and become granular, lose their nucleus, and are broken up into fragments. Successively the fungi invade greater numbers of vessels until the capillaries, the gland cells, etc., are filled with them, and carry in their track the work of destruction of the tissue. Along with the invasion is going on an intense inflammation, beginning in the portal and central veins, ending in enormous infiltration of the tissues with lymphoid cells and serous exudate. Farther increase of the infiltrate colliquates the decaying tissue, and an abscess results. The suppurative inflammation varies according to the manner in which the infection is propagated in the organ. When the connective tissue is the seat of the primary infection, then a number of lobules become gangrenous, and the abscess is formed secondarily. In wounds of the liver suppuration may begin the second day after the injury and abscess follow soon after. Primary abscesses are formed in consequence of wounds, pylephlebitis, peripylephlebitis, obstruction or total closure of the larger biliary ducts, with subsequent inflammation of the interlobular connective tissue. The liver—in *pyæmic abscess*—is usually of normal size, rather anæmic; in the tissues as well as under the peritoneal covering are scattered a number of abscesses of about 2 centimeters diameter, surrounded by hyperæmic tissue. A quantity of yellow and brownish matter, tinged with some blood, is found in the abscess. Here and there—near the larger abscesses chiefly—are good-sized portions of the tissue in a state of partial degeneracy and infiltration with fat. They are grayish yellow and show gradual softening and formation of abscesses. In abscess from pylephlebitis and compression of the liver cells there is found thrombosis of the portal vein and suppurative inflammation of the interlobular connective tissue, near the veins; great dilatation of some biliary ducts, innumerable miliary abscesses in the liver, inflammation of the splenic and mesenteric veins, abscesses in the brain, seropurulent peritonitis, swelling of the intestinal walls and follicular catarrh, ulcers in the duodenum and rectum, swelling of the mesenteric glands, tumors of the spleen, emphysema of both lungs. Fatty degeneration of the biliary ducts sometimes produces abscesses in the liver and are followed by perforating abscesses into other cavities, chiefly into the thoracic or abdominal cavity and formation of fibro-purulent, peritonitis, pericarditis, or pleuritis.

Besides transitory dilatations of the hepatic vessels there may be formed permanent vascular dilatation in consequence of inflammation of the parenchyma of the liver or from hæmostasis either in the cardio-pulmonic circulation or in the general circulation of the body. Occasionally the vascular lesion is confined to a limited part of the organ. The affected portions are remarkable by the dark color in the widened veins, or by paleness of the tissue, which is partly compressed. There is usually but little histological alteration to be noticed in this disease.

Diffuse Chronic Indurative Inflammation. Atrophic and Hypertrophic Cirrhosis of the Liver.

Chronic indurative inflammation is always insidious and slowly progressive. Very seldom is it the sequel of an acute process. The first stages consist of considerable infiltration of lymphoid cells in the interstitial tissue. The periportal connective tissue is chiefly, and the most early, involved part. Gradually the indurative process passes into the lobular parenchyma. As a rule the lesion is formed in spots; very seldom is it diffused over the whole organ. Subsequent to the infiltration is formation of connective tissue produced from large fibroblasts, that is, cells with very transparent nuclei. Steadily the disease progresses by accumulating lymphoid and fibroblast cells in the capillaries and eventually formed connective tissue. With this intravascular infiltrate are associated extravascular developments of connective tissue round the capillaries and between the lobular cell rows, which they at first compress and at last replace. Under such circumstances great quantities of parenchymatous cells are destroyed. The destruction of the cells is followed by pigmentation of the remaining tissue, from the debris of the blood in the destroyed capillaries. A portion of the pigment is derived from the bile, which stagnates in the obstructed biliary ducts. After the indurative process has been going on for some months the connective tissue becomes hyperplastic, which also is first found in the periportal connective tissue, to which it is sometimes permanently confined, or from which the hyperplasia progresses toward and into the lobular structures. The cellular groups are driven apart and separated from the adjacent capillaries. Some are destroyed, some remain and form wide biliary canals communicating with the still preserved biliary ducts. The biliary

TABLE IV.

FIGS. 1, 2, 3, 4 represent extraordinary dilatations and hypertrophy of the thoracic duct and groups of lymphatics, co-existing with enormous dilatations of many large arterial trunks in different portions of the body, reported by *Jules Cloquet* in *Pathologie Chirurgicale*, and drawn by *R. Carswell*. Fig. 1.—Aneurism of basilar artery. (A), cavity of aneurism; (B), portion of basilar artery; (C), the vertebral arteries. Fig. 2.—Portion of the brachial, ulnar, and radial arteries, extensively affected with aneurismal dilatation. The size of the tumors were different in the same vessel. (A), brachial artery laid open; (B), ulnar; (C), radial artery; (D D), aneurismal tumors projecting from the external surface of those arteries; (E E), aneurismal sacs communicating with the brachial. Fig. 3.—Varicose dilatations of the iliac arteries. These vessels and their chief branches presented a remarkable increase of bulk; flexuous and elongated they formed numerous sinuses and dilatations of various forms and dimensions. The walls of the dilated portions were soft, flaccid, and collapsed; their color paler than natural, the middle coat had lost its yellow tint, its circular fibers were less apparent. In some parts they were selerotic. (A), termination of abdominal aorta; (B), inferior mesenteric artery: (C), left common; (D), right common iliac arteries; (K K), sinuosities; (M M), dilatations. Fig. 4.—A portion of the thoracic duct, etc. (A), portion of the diaphragm; (B), thoracic duct; (C C), two large branches; (D D), lymphatics, some of which are drawn in outline.

FIG. 5.—Specimen of carcinoma confined to the mucous coat of the duodenum, with thickening of the muscular coat, and infiltration into the mesenteric glands. The mucous membrane (B B) is in some places an eighth of an inch thick and raised in a number of convolutions—like the surface of the brain—tinged with red, showing incipient hæmorrhage. At (D D) the primary stage of the disease is readily noticeable. In many parts the villosities are very large (E E) and prominent. Very many lacteals, arising from the mucous membrane (G G), and connected with the villi at (F). The glands are infiltrated with cerebriform cancerous matter and highly vascular. (A), mucous coat.

FIG. 6.—Tuberculous infiltration into the mesenteric glands. A portion of the ileum laid open and spread out, to which are attached a piece of the mesentery, several mesentric glands and lacteals. The tuberculous matter occupies the follicles of an enlarged agminated plaque (A). It also projects from the orifices of the solitary follicles (B). At (C) a less advanced stage of tuberculous infiltration, small, round, slightly conical elevations, of a straw color, or light gray, upon the mucous surface indicate the early stages. Ulceration of the solitary follicles and plaques of the mucous, muscular, and serous tunics of the intestine are indicated at (D E F) in consequence of the tuberculous deposit in the tissue. Ulceration of the mucous follicles seen at (D); ulcerations of the mucous, sub-mucous, muscular, and sub-peritoneal tissues are presented at (E F). The muscular coat is denuded and ulcerated. The sub-mucous tissue filled with miliary tubercles. The lacteals are dilated and filled with tuberculous matter (G); the mesenteric glands (H K L) are enlarged, some exceedingly, and all filled with tubercular masses. At (M) the lacteal communicates with an ulcerated plaque, passing beneath the atrophic mucous tissue.

ducts are usually not injured and may, on the contrary, increase in extent. *Ackermann* has injected into the newly-formed bile ducts from the hepatic duct. Circulation in the formed periportal connective tissue is considerable. However, many portal-vein branches are by the inflammatory process destroyed, but many interlobular branches remain intact. In the portal regions venous stasis often takes place, producing swelling of the spleen, ascetis, and very often statie hæmorrhage. The hepatic artery circulation is not nearly as much involved as the other circulation. Its branches dilate and many new arterioles are produced and furnish nutrition to the thickened capsule of *Glisson* as well as to the lobular structures. In atrophie indurative hepatitis the artery is injured and many of its branches obliterated. Not supplying the lobular cells with sufficient circulation they either decay or undergo fatty change.

In consequence of obstruction of the portal circulation within the liver many communicating veins with the portal-vein region of supply which, under ordinary circumstances, are so insignificant as to escape the notice of most anatomists, become gradually enormously dilated and assume unexpectedly large proportions. Such are some of the veinlets of the suspensory ligament of the liver lately observed and described by *Sappey*. The veins of the abdominal walls, of the diaphragm and æsophagus, also those which anastomose with some of the veins of the lower extremities enlarge to an extraordinary degree, become often very tortuous and dilate into many varicosities. The extent of the inflammatory process corresponds to the extent and intensity of the exciting cause. When the inflammation issues from the branches of the portal vein or of the hepatic artery, the extent of inflammation will be according to how many branches of the vessels are primarily involved; circumscribed when a few, more diffused when many of them are affected. But when the inflammation proceeds from the periportal connective tissue and expands into the interlobular spaces it will be very general. A peculiarity of the inflammation, when arising in the biliary ducts, is that it appears in the shape of globular, circumscribed centers, containing biliary coloring matter, situated partly in the lobules, partly in the periportal tissue. Biliary hepatitis is sometimes hyperplastic, sometimes pyogenie.

In recent inflammation of the liver there is always enlargement of the organ, which is the more considerable the more extensive the inflammatory process is. Large centers of inflammation show the unassisted eye grayish or greenish-colored spots (Section VII, Table I, Fig. 1, C C). When connective tissue is developed by hyperplasia, the organ will still further increase in size. In diffuse hepatitis the enlargement will be necessarily greatest. In appearance such an organ is very much like an infiltrated liver (Section VII, Table II, Figs. 5, 6). In the region of the portal-vein connective tissue and in the lobules there are accumulated lymphoid corpuscles and biliary pigment, also connective-tissue cells; the whole infiltrate being either grayish-red (Section VII, Table II, Figs. 3, 4, 5, 6), yellowish, or greenish, according to the amount of the coloring-matter of the bile or the blood. Similar appearances exist when there is retention of bile by enormous development of connective tissue in the interlobular spaces, by mechanical stagnation of bile, or obstruction within the gall ducts. The lobules are then also either brownish-red, brown, yellow, or gray (Section VII, Table II, Figs. 1, 2, 3, 4).

The quantity of such newly-developed connective tissue is sometimes very great, and the enlargement of the organ extraordinary (10 to 12, *ib.*). This condition has been called *hypertrophic induration or hypertrophic cirrhosis*. The surface of the liver is then smooth, the texture tough and dense. The lobular structure, according to the quantity of new tissue, is more or less obliterated. The parenchymatous tissue remains sometimes preserved to some slight extent. At other times it becomes so atrophied that the whole organ shrinks. In the atrophic stage the tissue is crossed by a greater or lesser number of *striæ* of grayish-red, yellow, or yellowish-green-colored connective tissue, in the meshes of which lie remnants of the parenchyma of the liver, having a reddish-brown, yellow, or gray color (Section VII, Table 1, Figs. 3, 4, 5). With the shrinkage of the connective tissue (which gradually follows), the whole liver becomes smaller, and often very small. Its surface then assumes an embossed appearance, the depressions formed of the shrunken connective tissue, the elevations of the remnants of the parenchyma (Section VII, Table I, Figs. 2, 3, 4, 5; Table II, Figs. 5, 6). The smaller the remnants the smaller will be the elevations, and the more abundant the depressions between them. The several appearances produced thereby have received the names of granular, lobular, and *lobar cirrhotic* atrophy; the last also passes as *Laenec's cirrhosis*. Of late years, chronic interstitial hepatitis has been the subject of very numerous researches and of as numerous experiments, which have led to attempts at subdivision of the disease into many classes, but which were really of no practical value. At present the English, French, and some American pathologists assume two main forms (advocated by *Charcot* and *Gombault*), viz: *biliary cirrhosis*, having a hypertrophic character, and *vascular*, with subsequent atrophy. The Germans contend for two stages of the same disease, the primary or hypertrophic, and the secondary or *second stage*, as the atrophic. Both may have their origin in the biliary or vascular apparatus, or in the perivascular or interlobular connective tissue. Jaundice, which is sometimes very prominent, and at other times but very slight or nearly imperceptible in cirrhosis, does not seem to be directly connected with either the one or the other form or stage of the disease.

Syphilitic Hepatitis.

Acquired syphilis may give rise to a form of hepatitis which very much resembles, anatomically, the described forms of cirrhosis. Only concomitant symptoms in other parts of the body can furnish means of differential diagnosis of syphilis and cirrhosis. Usually *syphilitic interstitial* hepatitis is not diffused throughout the organ, but, as a rule, affects limited portions. The surface of the liver is generally uneven, as in cirrhosis, most frequently near the suspensory ligament. It has, in places, the appearance of cicatricial contractions, and the covering membrane is thick and wrinkled (Section VII, Table 2, Figs. 5, 6). A section upon a contracted spot shows accumulation of connective tissue, which spreads from a common center in different directions in a radiary form. This constitutes a gummatous syphiloma of the liver. The parenchyma of the organ between these cicatricial *striæ* is in an atrophic condition, of brown color, the lobules very minute. The connective tissue lodges very small caseous nodules of a gray color, somewhat diaphanous. Similar *gummata* are found in the organ itself, very often in great numbers. Many such cicatrices produce upon the surface of the liver a lobulated arrangement. The center of the *gumma* consists of *homogeneous*, *aneuclear*, decaying tissue, or granular detritus with some decaying cells; the histological character showing that of atrophied liver structure and infiltrated lymphoid cells, which never developed into connective tissue.

This destructive process in the early stages of the inflammation shows the total inability of the parenchyma to become regenerated, or even to cause development of the neoplasm into living tissue. The cells die from lack of nutrition, because the syphilitic virus destroys the vascular structure, and the circulation ceases.

In hereditary syphilis similar infiltrations and minute gummata are found in the liver, especially of new-born babes, or those dying in utero in very early fœtal life. These infiltrates are all of microscopic size and can not be seen by the unassisted eye. Indurative syphilis is common in both kinds and are often associated with enlargement of the organ. The color of the liver is, in such conditions, red, yellow, gray, or speckled according to the nature of the infiltrate and the intensity of the tinge it assumed. (*Gubler.*) Very often vast numbers of miliary gummata are diffused through the liver, producing innumerable small gangrenous spots which nearly destroy the function of the organ altogether.

Hepatic tuberculosis is either a partial lesion of general systemic miliary tuberculosis, or a *local* tubercular lesion. In the first case the tubercles are of miliary size, diffused in the periportal and acinous structures; in the second they form larger caseous and ulcerating focal deposits in all its tissues. There is always formation of hyperplasia, which encloses the tuberculous masses.

ENLARGEMENT OF THE LYMPHATICS.

MESENTERIC TUBERCULOSIS.

Fig. 6.

Fig. 4

Fig. 5.

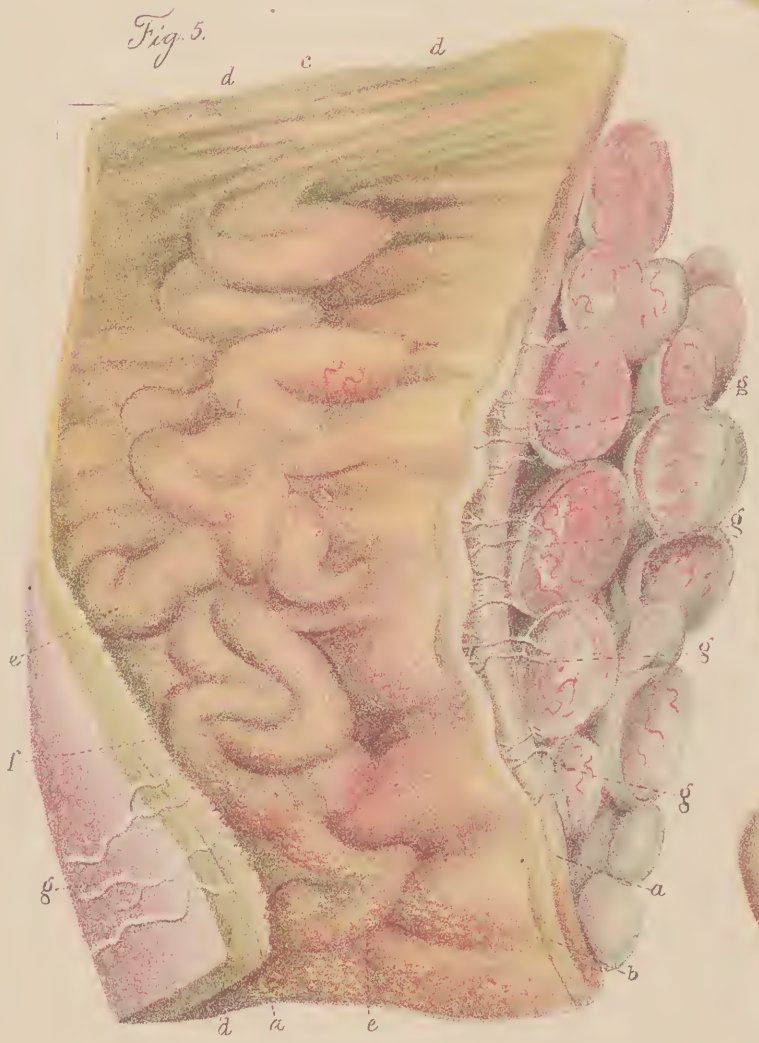
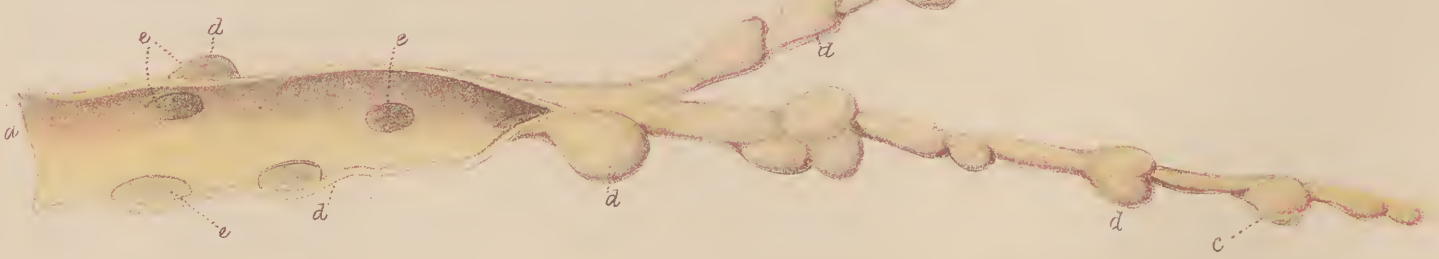


Fig. 3

Fig. 1.

Fig. 2.



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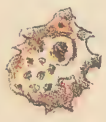
CIRRHOSIS OF THE LIVER.

Sec. VII. *Tab. V.*

Fig. 1.

Fig. 2.



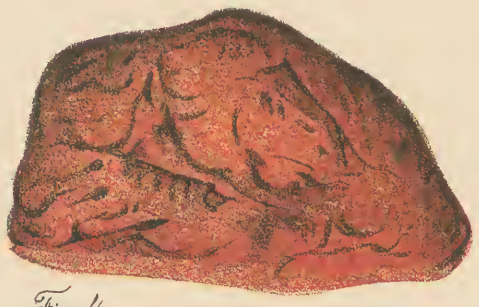
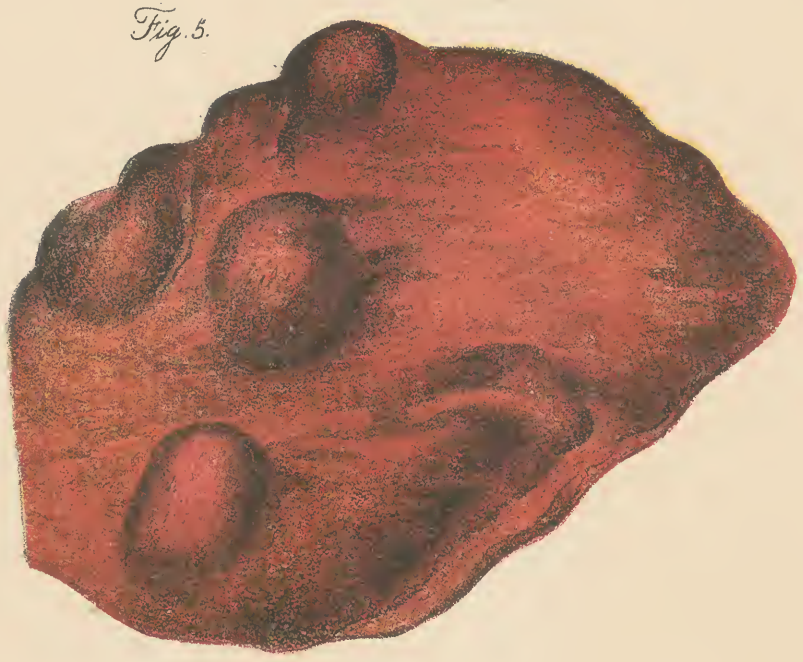


Fig. 4.

Fig. 5.



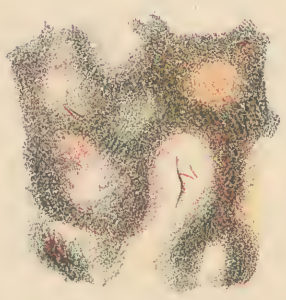


Fig. 3

Fig. 6.

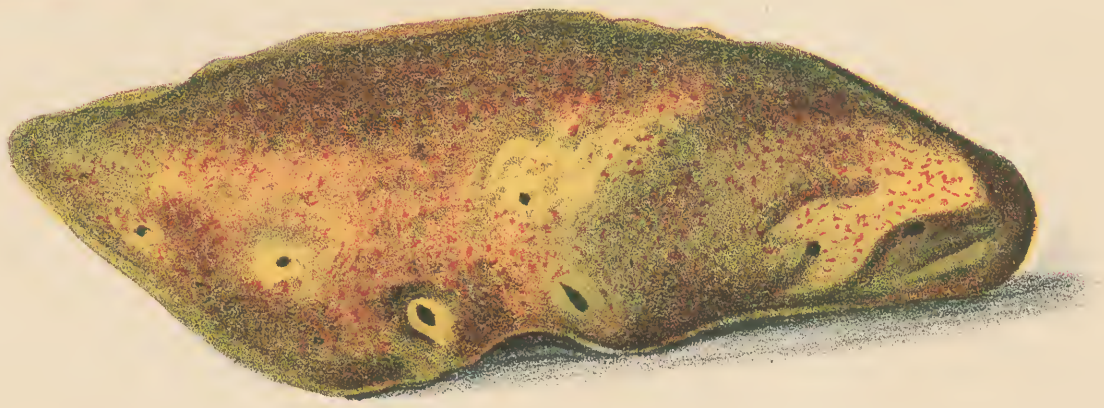


Fig. 6. a.

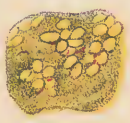


Fig. 6. b.

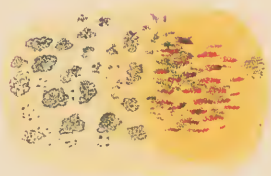


Fig. 6. c.

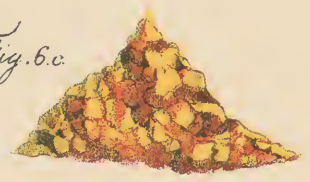


Fig. 7.

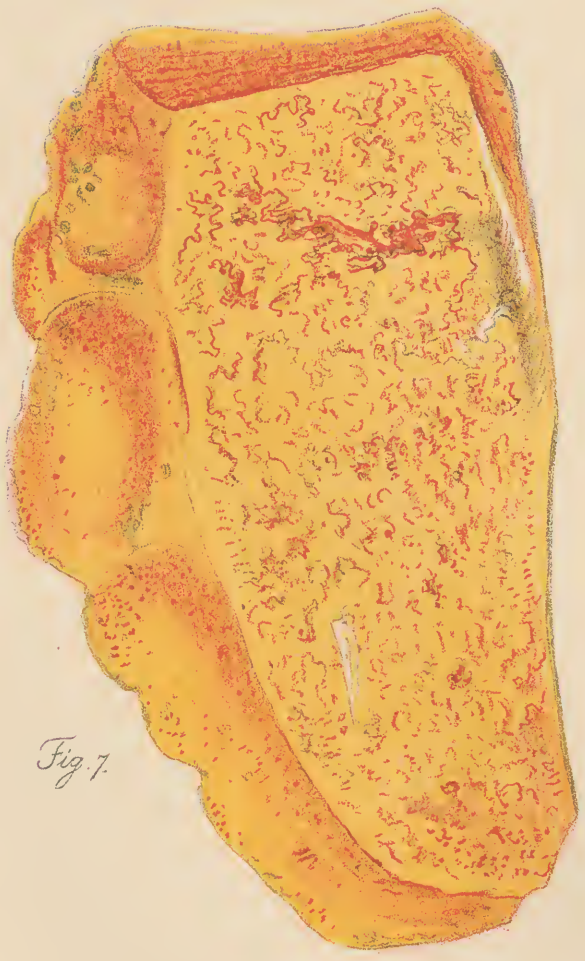


Fig. 8.

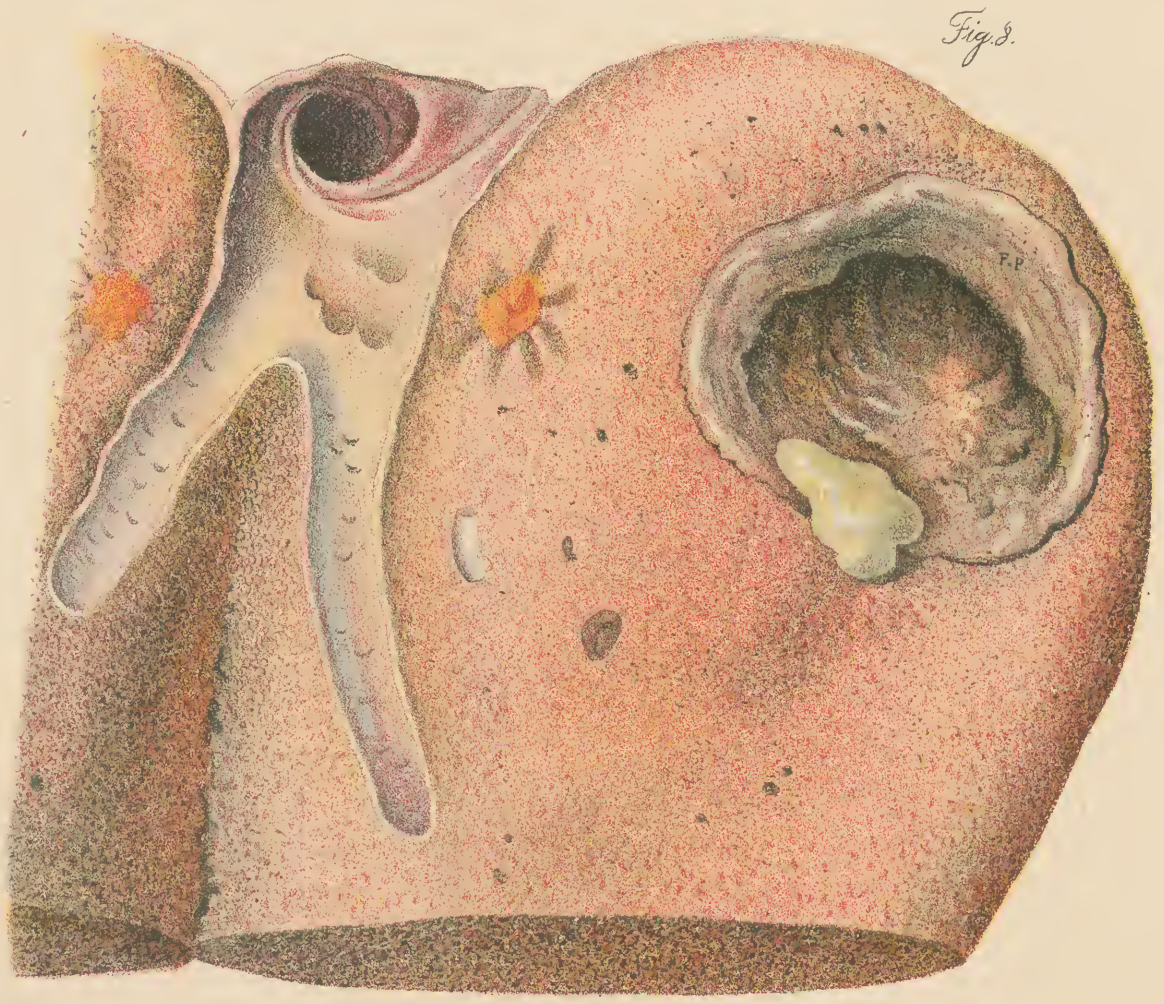


TABLE V.

FIG. 1.—*Cirrhosis of the Liver.*

CASE.—A man forty-five years of age. Comes of a family affected with liver diseases. Has worked very hard, and lived on scanty fare, and in dark, damp rooms. Suffered for some years from dyspepsia and constipation. Is very irritable. Present condition: Abdomen very tympanitic, but not hard. Liver accessible to palpation; feels indurated, with uneven upper surface; extends far into the left hypochondrium. Urine brown color; specific gravity, 1022.0; leaves a dark-brown deposit. Fæces gray and hard. Hæmorrhoidal tumors in rectum. Pulse slow and feeble. Steady cough. Died from exhaustive gastro-enteric hæmorrhages. Autopsy shortly after death: A quantity of brown serum in abdominal cavity, containing much albumen. Intestines distended with gas. Gastro-duodenal mucous membrane very red; cavities filled with blood. Cause of hæmorrhages, static venous congestions in portal vein and obliteration of many branches of mesenteric veins. Portal circulation in the liver nearly destroyed. Part of the larger lobe occupies nearly the whole transverse diameter of the abdomen, is of a grayish-yellow color, and filled with granular indurations. The tissues are utterly anæmic, the vessels nearly obliterated. The biliary capillaries are partly changed into fatty tissue, the liver cells unrecognizable.

FIG. 2.—Slightly magnified portion of liver, showing a lobule with the central vein.

FIG. 3.—Infiltration with fat molecules, much enlarged, showing total absence of lobular structure.

FIG. 4.—Nutmeg liver of another individual, who was a hard drinker. The alternating dark and light parts indicate a greater or lesser quantity of pigmentation of the tissue.

FIG. 5.—Hobnail liver, enormous tumors on the upper surface. The peritoneal covering and the capsule very thick and fibrous. The organ, hard and tough, shows a chronic state of interstitial hyperplastic inflammation.

FIG. 6.—Infiltration with fat and coloring matter, in enteric typhoid. The organ almost of normal size, soft, and shines on a cut surface. Circulation diminished, but the vessels nearly normal. A number of places in the organ thoroughly infiltrated with fat and biliary pigment. Figs. 6*a*, 6*b*, and 6*c* show magnified fat and pigmentary molecules.

FIG. 7.—Represents a portion of a liver of a nineteen-year-old girl who died of anasarca. The right lung was normal; the left adherent to the ribs, and a cavity in the apex filled with pus. A few caseous tubercles scattered here and there. The heart was small and soft; the liver small and soft, with many elevations on the surface, penetrated with countless numbers of granules, consisting of fat and bright-yellow pigment. The liver cells were wiped out. Kidneys of normal size, but pale and soft. No albumen in the urine. Liver capsule very thick.

FIG. 8.—A considerable encysted abscess in the thickest portion of the right lobe, near the posterior border. The cyst (F P) is ingrown with the liver tissue, and can not be enucleated. By the side of the suppurating portion of the abscess, near one of the branches of the hepatic vein, there is an orange-colored spot, which seems to be a cicatrice of an old abscess. The remainder of the liver tissue is nearly normal.

In the caseous portions gangrene takes place, leaving behind cavities filled with debris of the sphacelus stained with biliary pigment.

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ON TUBERCULOSIS OF THE LIVER.—Arnold; Orth.

Tumors.

Non-malignant and malignant neoplasmata are not uncommon in the liver, especially carcinomatous formations and hyperplastic infiltrations in the glandular structures, which often compress the gland cells and produce atrophy and subsequent destruction of the whole organ. They are usually of metastatic origin, although they may also be caused by local inflammations, etc. The functional disturbances consequent upon such tumors and other neoplasmata depend on the kind and the extent of the anatomical alterations in the organ.

Functional Disturbances of the Liver and their Clinical Character.

The specific functions of the several structures of the liver not having as yet been definitely ascertained, its different secretions must be considered as derived from the whole organ. The abnormal conditions in the quantity and quality of those secretions, as manifested in many diseases of the organ, form the chief clinical features of the functional disturbances. There may be a partial or total cessation of the flow of bile from the liver into the intestine from simple mechanical obstacles in the biliary passages, while the secretion of that substance may go on in a more or less regular manner for some time in the organ. Its elimination will take place by the hepatic vein, and icterus will follow, in a graver or lighter form in the body.

As the biliary acids thus carried into the blood are capable of dissolving the blood corpuscles, a train of disturbances of the most important functions of the body will, of course, follow, which will vary in intensity according to the quantity of the biliary poisons and their longer or shorter action. Without healthy blood corpuscles absorption of oxygen during respiration can not take place. Without sufficient quantities of oxygen in the blood no brain activity can exist; no muscular tonicity can be maintained in the body for any length of time without central nervous activity. Respiration becomes superficial and inadequate; the weakened heart, although at first making a supreme effort to overcome the effect of the more or less stagnant blood in the vessels, loses its power more and more. In measure as respiration is diminished, the blood pressure—which, in the early stages of *pernicious* jaundice exists, and assists in driving out the enormous quantities of destroyed blood corpuscles through the kidneys, in the form of *hæmoglobin-uria*—becomes more and more reduced. The body is in a state of complete prostration. The bile from the sluggish current of the blood is deposited in the tissues, which turn yellow from the coloring matter, and great quantities of fat collect in the parenchymatous organs, the effect of fatty change of the cells produced by the biliary acids. The stomach takes up biliary salts from the blood, and insupportable nausea follows, with fainting, and often gastric hæmorrhages. No digestion can take place. In fact, there is usually a state of total anorexia and utter disgust for food. The urine becomes scantier in measure as the blood pressure sinks, although great quantities of water are often drunk to satisfy the unquenchable thirst, but they are usually speedily vomited up, or passed through the swollen intestines as a serous diarrhœa. Death generally supervenes from asphyxia. In lighter forms of retention the symptoms are, of course, much milder, and many are absent altogether.

Suppression of secretion of bile, *acholia*, being usually a sequel of destruction and alteration of the cholepoetic apparatus, is characterized by great disturbance of digestion, with lack of appetite, anorexia in the latter stages, and many disturbances in the abdominal circulation (the latter are due to disturbed hepatic circulation); constipation; nervous prostration; often renal diseases; usually ascites, or even anasarca (but which is also really caused by disturbance of thoracic and abdominal circulation, which has brought on the disease of the liver and *acholia*).

In ordinary jaundice, without closure of the ductus choledochus, the yellow color is due to absorption of biliary coloring matter only, for biliary acids are generally absent in the tissues, and even in the blood. Polycholia, or hypersecretion of bile, is really a *myth*. Periodical collections of much bile in the gall-bladder, with sudden discharge of the secretion into the intestine and its evacuation by the stomach or intestine, are frequent enough in many cases of gastro-duodenal catarrh, in chronic catarrh of the cystic and hepatic ducts, and in many cases of liver troubles. Abnormal composition of bile, when several of its components are absent or in insufficient quantities, but with admixture of much mucus from the biliary ducts or from the intestines, is not uncommon, yet such a condition can not be considered due to hypersecretion of bile, but to temporary retention followed by sudden discharge.

That glycogen is produced in the liver has been proved not only by its discoverer, *Claude Bernard*, and shortly afterward by *Hensen*, but since by countless experiments and observations. Its role in the bodily economy is not positively known. The older physiologists (*Pavy* and others) took it to be a sort of bodily combustible, producing heat when consumed in the tissues. *Claude Bernard* and his school, to which many even at present still belong, thought that it supplied the young cells in their formation with glucose, a process analogous to that of cell nutrition in vegetables. *Naunyn* (Arch. f. Exper. Pathol.) and other modern investigators have shown the error of this opinion in the very latest classic researches of *Claude Bernard* himself. They found that at the time of greatest cell formation in the embryo no glycogen was present, or at least in very minute quantities, but that it abundantly existed in the livers of older fœtuses after the tissues were formed, and that only in places where no new cells were forming in the placenta could glycogen be found (*Naunyn*). In a pyogenic membrane on the plural surface, with enormous exudation of pus, the same author found a mere trace of glycogen. Many more examples could be adduced to show that glycogen is not always connected with formation of cells. This substance is a derivative of retrogressive metamorphosis going on in all the tissues of the body. In the muscles, where great chemical changes take place, a variety of glycogen is always found, *achrooglycogen* (*Bohmer* and *Hoffman*). It is formed by the serum of the blood from *xantoglycogen*, when this is forming sugar, by hydration, in the blood and tissues. In the liver it is formed both of albuminates and carbo-hydrates—sugar, glycerine—by abstraction of the elements of water. As it is capable

TABLE VI.

FIGS. 1, 2.—*Hyperplastic and Indurative Splenitis.*

FIG. 1.—*Case:* A woman of forty years, who died of anasarca. The lungs, in which a number of small tubercles existed, adhered to the ribs. A large cavity in the left upper lobe. Heart almost normal. Kidneys little affected. Liver undergoing fatty metamorphosis, usual in pulmonary consumption. The spleen (Fig. 1) was 120 millimetres long, 84 millimetres wide. One-third of the parenchyma in a state of inflammation. The capsule over the inflamed parts thick. The inflamed tissue very hard, and filled with fibroid exudation mixed with fat molecules. Its color whitish yellow. Contains very many lymphoid bodies. (A), surface of the spleen. (B), inflamed part. (C), surface of a section of healthy part. (D), locality of fibroid exudate.

FIG. 2.—*Indurated spleen* of a man who died of anasarca. The heart was small, the lungs œdematous. The liver very small, with imprints of ribs upon its surface. The kidneys had undergone fatty changes. Albuminous urine in last illness. Spleen enlarged, 200 millimetres long, 84 millimetres broad. Very dense, and cuts like hard cheese. Is of red color. Veins contain some blood. Surface of section shows neither capillaries nor malpighian bodies, but only a mass of dense fibroid structure.

FIG. 4.—Enlarged spleen. Weight, nearly nine pounds, avoirdupois. In the red parenchyma are broad, nearly spherical projections, and some pale granulations containing gray-colored and dark malpighian bodies, and a quantity of grayish, amorphous substance. A number of exceedingly enlarged and deformed follicles, forming an indefinitely-shaped mass, project above the cut surface. Portions of the mass are of different colors. (A), pus and black pigment, The vessels are filled with black thrombi (C). Pseudo-membraneous deposits on the thickened capsule (C).

FIGS. 3, 3*a*, 5, 6.—*Condition of the Spleen and Marrow Tissue in Typhus Recurrens.* (*Ponfick, Virch. Arch., Vol. LXII, p.* 154.)

FIG. 3.—Spleen, 15 centimetres long, 12 centimeters wide; weight, 330 grammes (about 10 ounces); 5 centimetres thick. Outer surface represents a much tumefied, dense structure, constituting about a third part of the organ. A transverse section shows in the lower portion a number of bright yellow dry spots, surrounded by bluish-red, slightly-swollen parenchyma. The upper portion has a number of yellowish spots of a smooth, glossy appearance on a cut surface. The balance of the parenchyma has a deep bluish-red color, and is full of blood. The follicles are gray.

FIG. 3*a*.—*Spleen of Another Person Similarly Affected.* Pulpa of a bright purple color, and soft; follicles gray and not very prominent. Trabeculæ indistinct. On upper extremity of the organ a number of yellowish-white miliary infiltrates having dark central depressions.

FIGS. 5, 6.—*Morbid Alterations in the Long Bones of an Individual who Died of Typhus Recurrens.*

FIG. 5.—Upper extremity of right tibia. A longitudinal section of the epiphysis shows a triangular spot of a grayish-green color. The center of the spot is dark, and is surrounded by lighter-colored alterations, marking several stages of the necrotic processes. The darker portion contains more pigmentary matter, and detritus of more advanced changes of the marrow and cancellous tissue.

FIG. 6.—Middle portion of right femur of another person alike affected. A longitudinal section has on its cut surface white spots and stripes of uniform opaque and tallow-like density. The surrounding marrow is softer, and is in a state of hyperæmia. All other long bones are in a similar hyperæmic state.

of readily dissolving blood-coloring matter, it either serves to dissolve and eliminate the debris of blood, carried in the portal vein, with the bile, or at least assists in so doing (*Schopper*, Arch. f. Exper. Pathol.) Its role in the muscles may *possibly* be that of dissolving the hæmoglobin steadily found in the muscular veins after muscular exertions.

Glycosuria, *diabetes milletus*, is very often of hepatic origin, and may be considered as a disturbance of function and not a disease of the liver. This disease will be considered in the part of this work where diseases of the kidneys will form the subject.

Derangement of the portal circulation, especially when gradually produced, interferes but little with the function of the liver, although extensive and often grave disturbances in the intestines are caused thereby. Inflammations of the portal vein, with thrombosis, may sometimes be followed by abscess of the liver. Ascites and secondary disturbances are always produced by phlebitis and periphlebitis of that vein. It often terminates fatally.

Derangements in the hepatic artery produce great disturbance in the circulation and consequent anomalous nutrition; usually gangrene of the liver structure—for that vessel constitutes *the* nutrient vessel *of all* tissues in that organ. It is, however, supplied with so many collateral currents that in man such occurrence is of the greatest rarity.

The causes of diseases of the liver are many, and their ætiology may very often be that of very many infectious diseases of the whole body, as well as poisons of many kinds, not less than climatic and local effects. In all high-graded inflammations taking place in the body, if not diseases, at least deep disturbances of the hepatic function will always exist. It is no wonder, as the liver is physiologically as well as anatomically connected with the organs of digestion, respiration, circulation, and elimination, and whenever any deep alteration takes place in those organs the liver must invariably be affected.

According to *Litten* (*Zeitschrift f. Klin. Med. IV*, pages 55, 70), three different kinds of morbid alterations in the retinal structures, in the fundus of the eye, are observed in diseases of the liver:

1. Extravasations in the fibrous layer of the optic nerve, near the papilla, or in the peripheral portions of the retina. They take place in congestive *icterus*, carcinoma in the biliary ducts, acute yellow atrophy, abscess, dropsy of the gall-bladder; in poisoning with phosphorus; in cirrhosis, both atrophic and hypertrophic forms, and in bilious pneumonia. He considers these extravasations as part of a general hæmorrhagic condition of the body, but not produced by biliary infiltration.

2. Various-sized, irregular, grayish-white spots on the retina of both eyes, found in poisoning with phosphorus (*pyrogallic acid, Ref.*), with subsequent atrophy and excessive fatty degeneration of the liver. These gray spots are fat molecules deposited in the retinal structures and in its capillary walls.

3. Typical pigmentary degeneration of the retina in atrophic cirrhosis, sometimes perceptible before any other manifestations of hepatic disorders. *Hemeralopia* is found in both the atrophic and hypertrophic forms of cirrhosis of the liver, without any perceptible change in the retinal structures. It is due to *intraoccular* increased blood pressure. *Carrillon* observed the fundus of the eye with the ophthalmoscope in many cases of various hepatic diseases, and found that the hemeralopia was due to disturbance of the intraoccular circulation, manifested by extremely thin arteries, varicose veins, and papillary œdema (*Progres. Medic.*, June, 1881). Ophthalmoscopic examinations of the fundus will very often, even at a very early period of diseases of the organs of circulation (the heart, vessels, hæmatic glands), indicate the presence of more or less disturbance in the parenchymatous organs, long before any other perceptible objective symptoms appear.

Of physical symptoms of liver diseases, its changed secretions and the disturbances consequent thereupon are often the most valuable. Icterus in all forms invariably shows disturbance of the liver function, which may have taken place either primarily in the liver or in the biliary ducts, and originating in intestinal disturbance. Inspection, palpation, and percussion are always necessary, usually indispensable, in hepatic diseases, and will be described in the diagnostic part of this work, with the nature of other physical symptoms.

Diseases of the Spleen.

The spleen is the largest lymph apparatus of the body and is both anatomically and physiologically connected with the chylopoetic organs. Its general structure is essentially that of lymph glands generally. Its parts consist of three anatomically-different structures: the capsule, the pulpa, and the malpighian bodies. The capsule is made up of sero-fibrous tough and dense connective tissue, with some few organic muscular fibers. From the capsule innumerable finer and coarser bands and strings pass into the pulpous structure in every direction, and form an inextricable net in the length and breadth of the organ, forming quite a solid framework for the rest of the pulpa tissue, made up of vast ramifications of the splenic arteries and veins and their accompanying lymphatics. Very numerous nerves, derived from several sources, enter into its tissues. Within the meshes of the dense network are found vast quantities of lymphoid bodies, larger nucleated cells, and many blood corpuscles, either in a state of complete preservation or in a more or less decayed condition. Blood pigment and other detritus, and young blood cells are also abundant in the splenic pulpa.

From the hilus the arteries, which are very large compared to the size of the spleen and very thick-walled, pass into the organ side by side with the very wide but very thin-walled veins, inclosed in a common sheath, surrounded by perivascular structure identical with that of the capsule. The vessels divide into branches at their entrance and traverse the organ in separate divisions, which never anastomose. The veins and arteries run parallel with each other until they reach their tertiary subdivisions, and then part from each other. The smaller arterial branches suddenly divide into very numerous slender and long-stretched branchlets, which also remain separate, forming terminal vessels. At the points of division and on the terminal course of the finest arteries, sacular enlargements on the outer coats are to be seen. They have received the name of malpighian capsules or follicles. They stand in immediate connection with the perivascular tissue. The veins, after separating from the arteries, also constantly subdivide, and terminate in venous capillaries. The perivascular sheaths continue along the whole course of the vessels, enfolding the arteries very tightly, but inclosing the veins rather loosely. At first this tissue is very thick and dense, but it becomes more attenuated when it reaches the smallest arterioles, and at last forms but a very thin fibrous layer upon the much-dilated terminal branchlets of the veins. The framework of the pulpa is made up of bands and strings which the capsule sends into the organ, and extends in every direction. Secondary and tertiary fibers spring from them, and, communicating with one another by finer or coarser bundles or single threads, form an inextricable network. From the perivascular tissue similar threads and strings are sent forth to still farther increase the intricacy of this net-like frame. In the innumerable meshes vast quantities of lymphoid corpuscles are imbedded in a soft, albuminoid cement. On the mode of junction of the arterial and venous currents in the spleen, authorities still differ.

Billroth, *Frey*, *Schweiger-Seidel*, and *Koellicker* contend (from their own observations) that the arterial capillaries at once terminate in the venous, without any intermediary capillary network. *Axel-Key* and *Stieda* contend that there exists a zone of extremely fine capillaries, forming communications between the arterial and venous capillaries. *Stieda*, *W. Muller*, and *Gray* claim that there are really no walled vessels between the two classes of capillaries, but that the arterial blood passes directly between the parenchymatous cells and their reticular threads, and into the smaller veinlets. Such glaring contradictions of perfectly competent authorities seem due to the difference of structural arrangements in the spleens of different animals experimented upon, and very

HYPERPLASTIC AND INDURATIVE SPLENITIS.

Fig. 1.

Fig. 2.

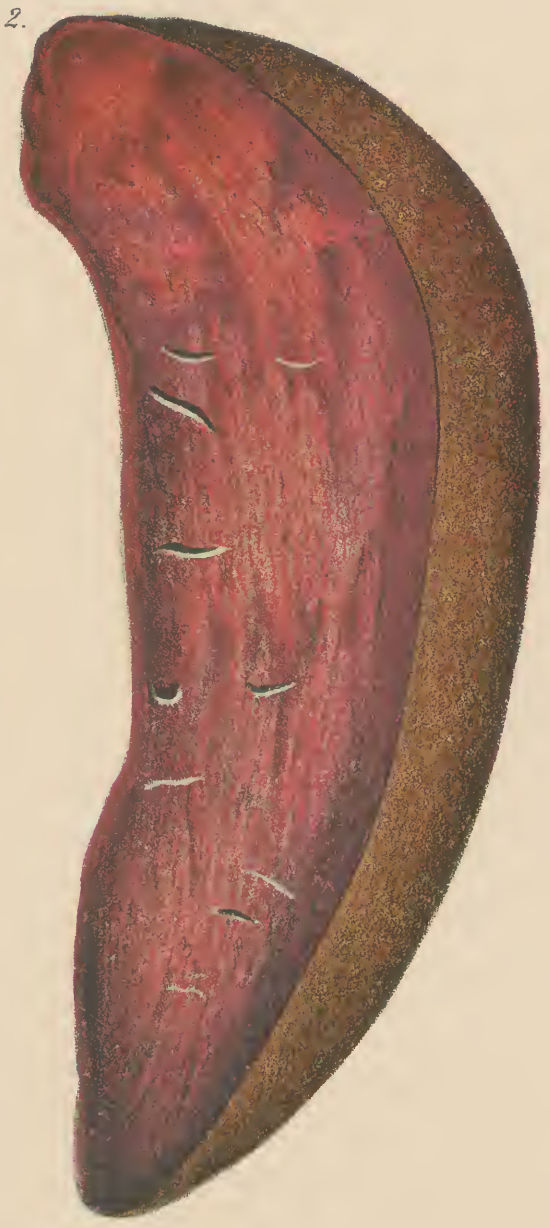

Fig. 4.

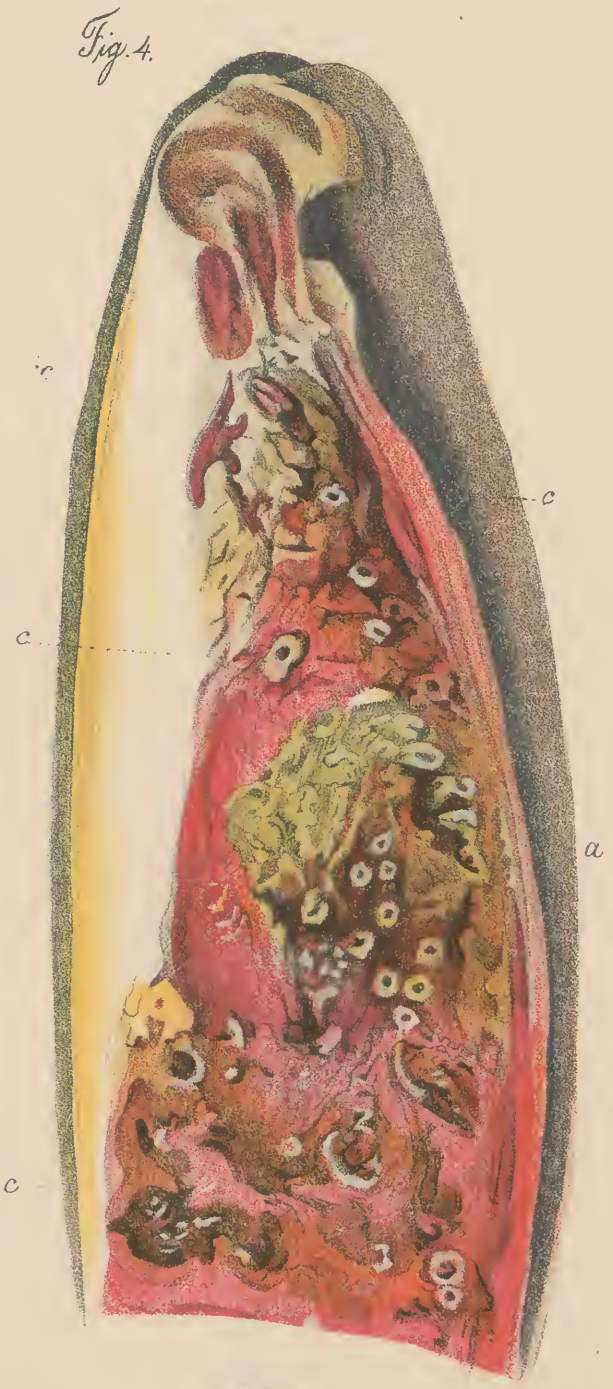

Fig. 5.

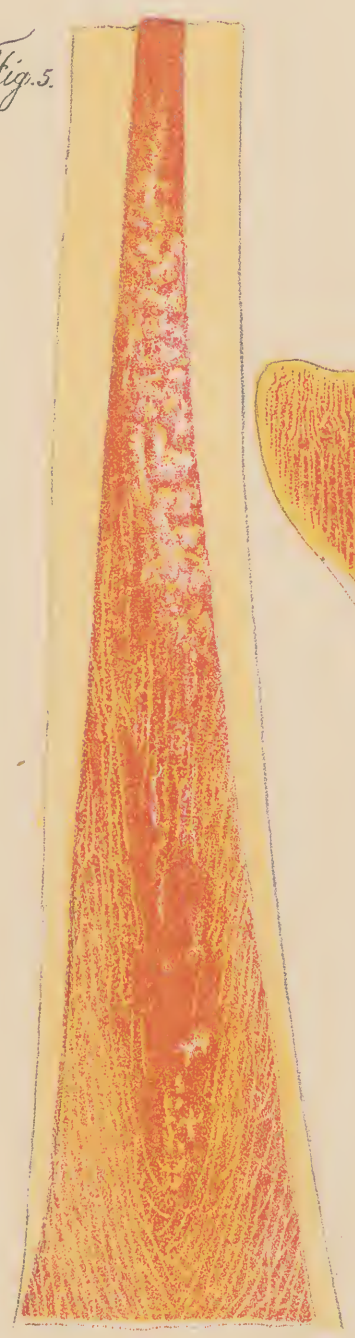

Fig. 6.

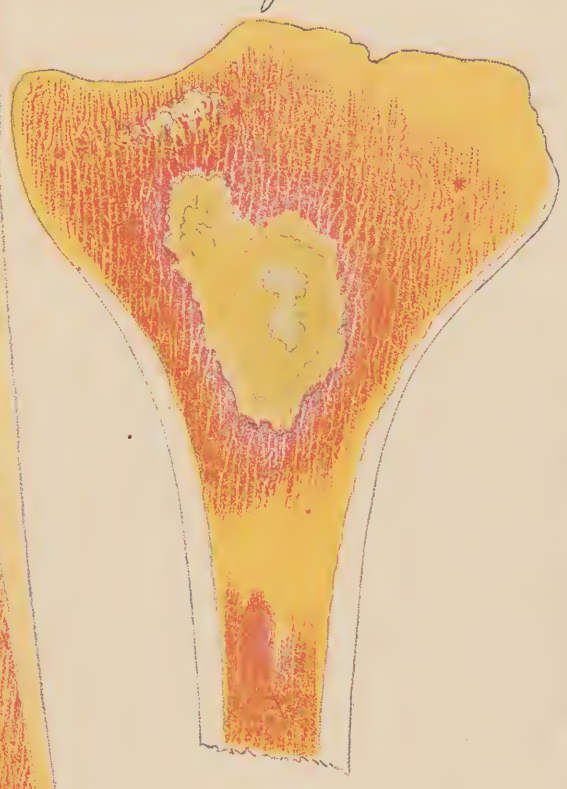

Fig. 3.

Fig. 3. a.

Sec. VII. *Tab. VII.*

ACUTE SPLENITIS, WITH FORMATION OF ABCESSES.

SOFTENING OF THE SPLEEN.

Fig. 1.

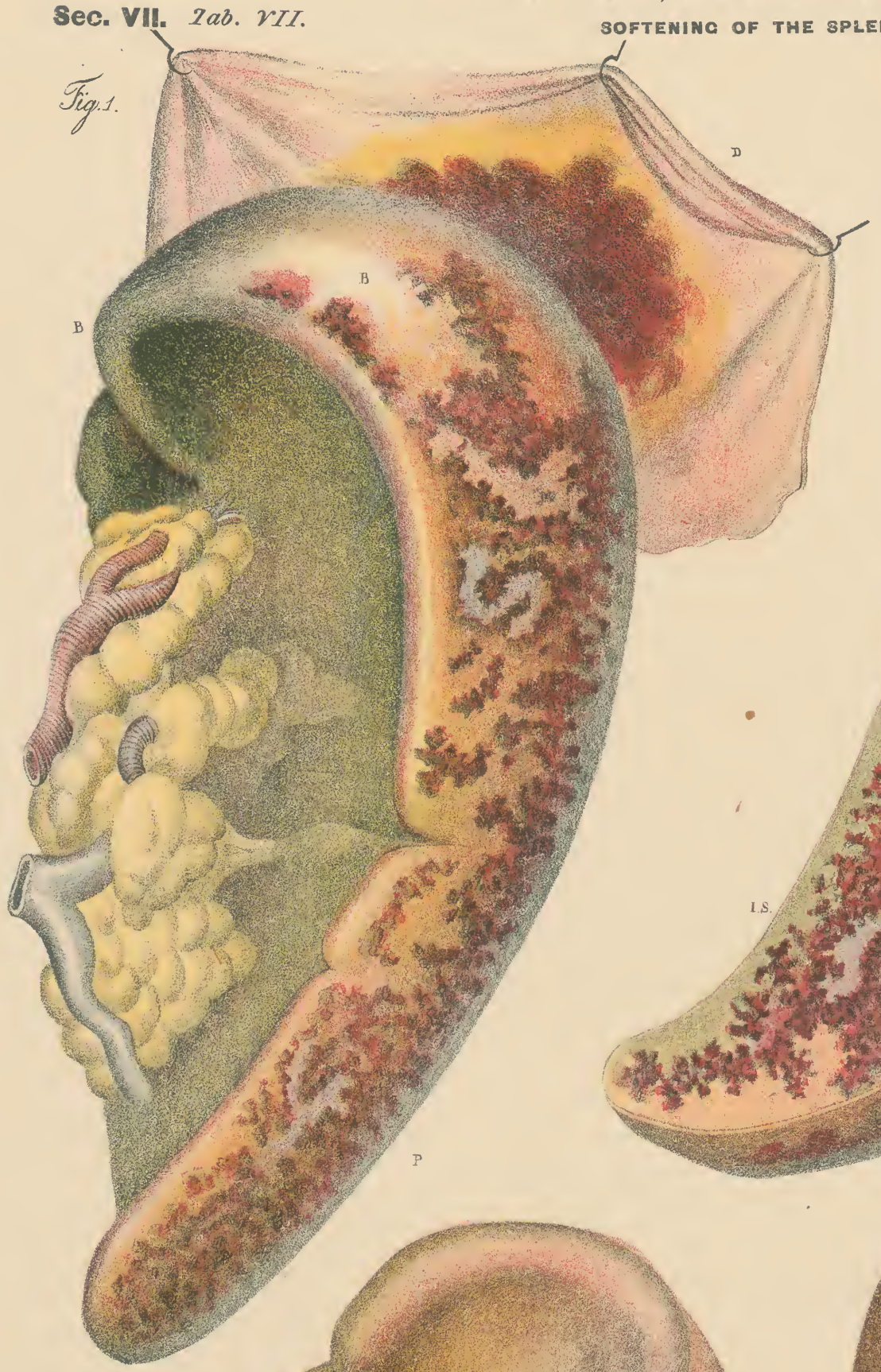

Fig. 2.

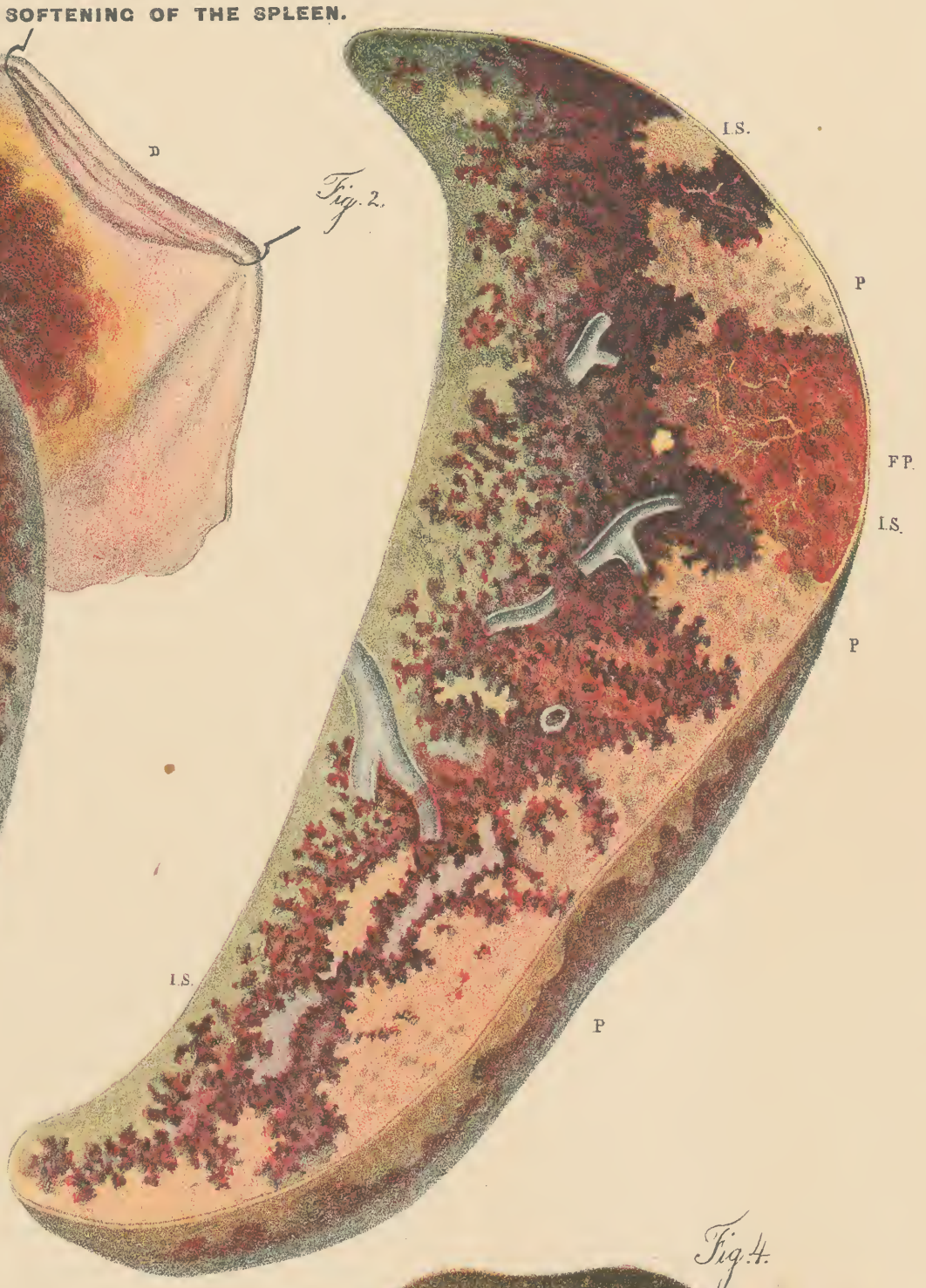

Fig. 3.

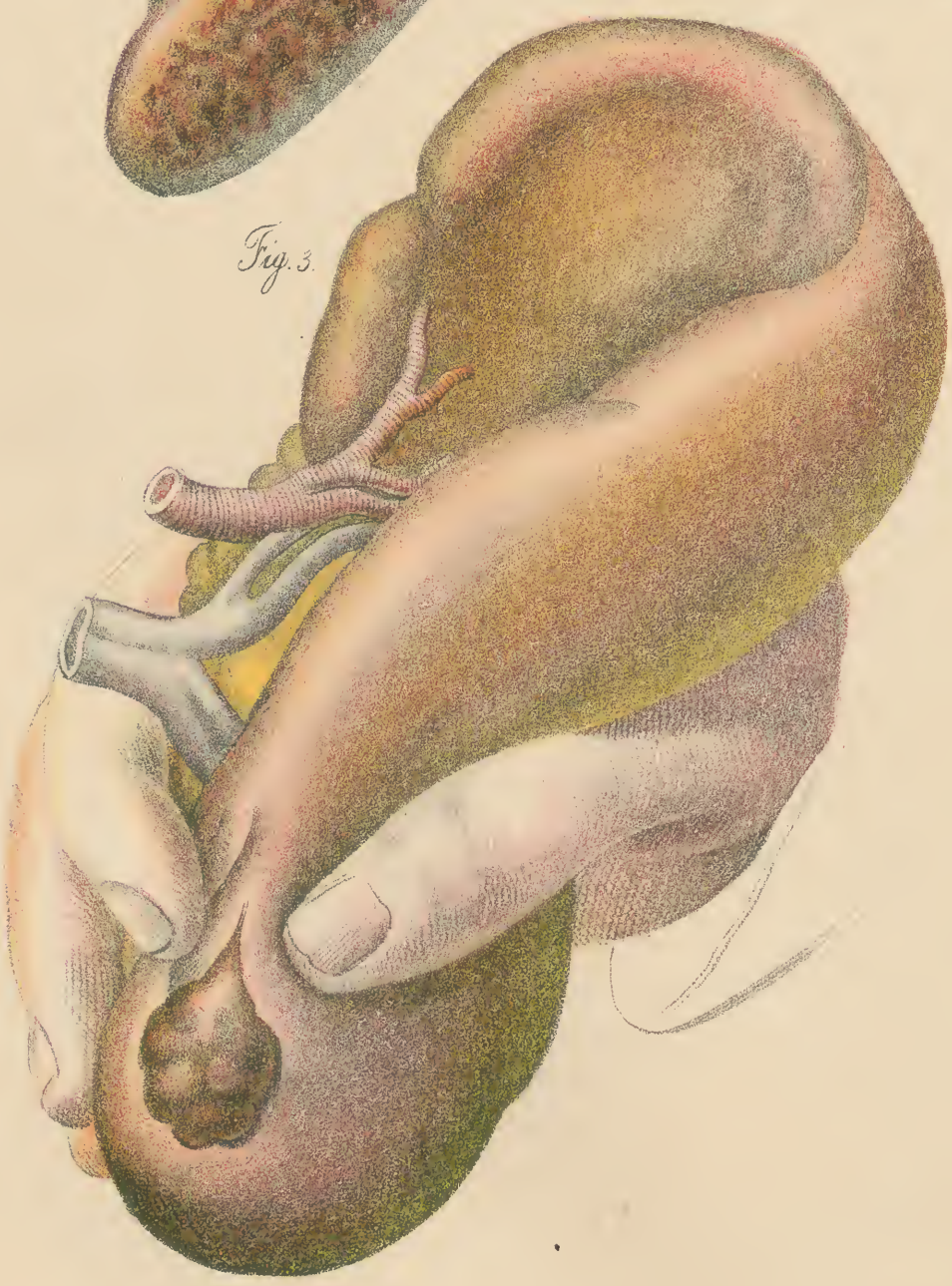

Fig. 4.

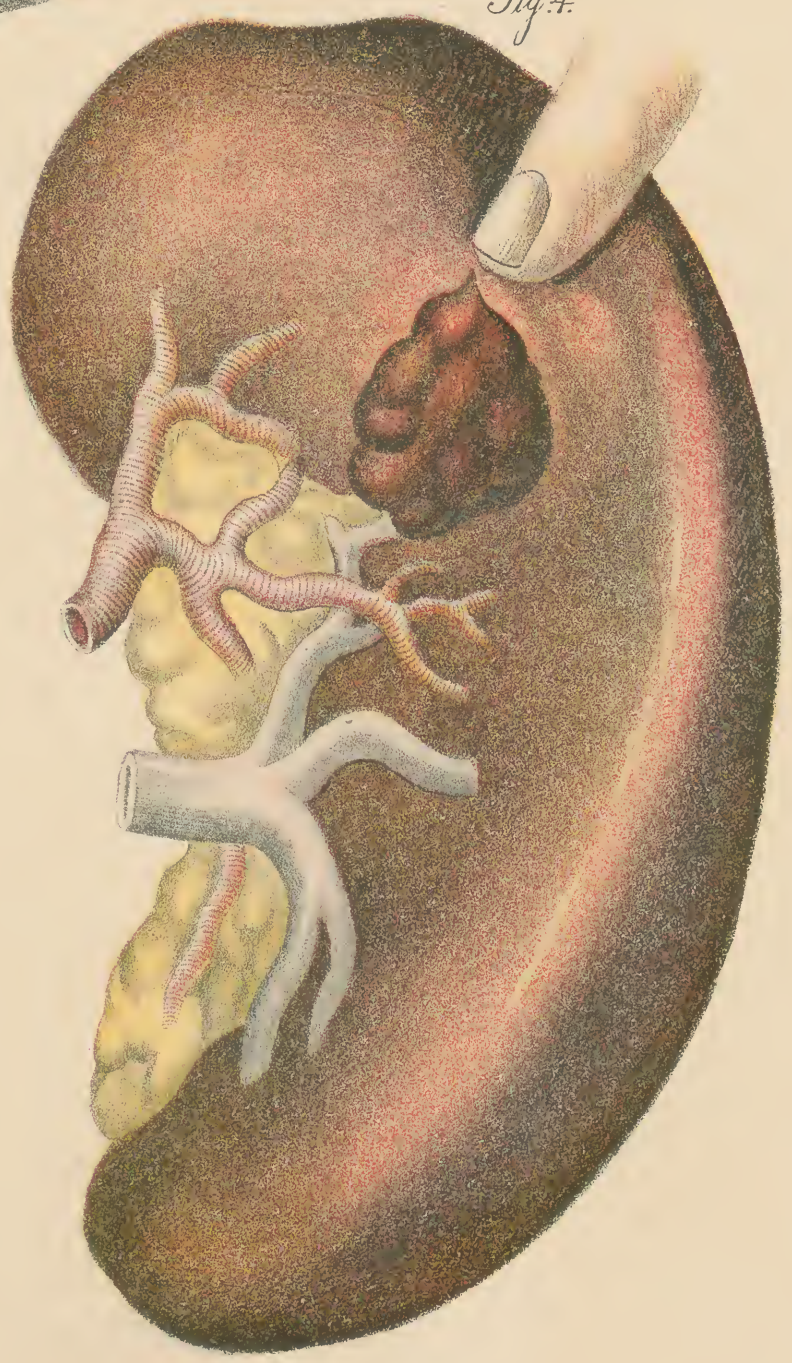

TABLE VII.

FIGS. 1, 2.—*Acute Splenitis, with Formation of Abscesses in its Tissues. Pernicious Intermittent.*

CASE.—A woman of twenty-eight years, of robust constitution, and strongly built. She was suddenly seized with a violent pain in the left shoulder. At first she experienced neither chill nor any rigor. Her tongue was clean, smooth, and moist, but very red; the pulse moderately frequent. Her face intensely red. No pain anywhere except in the shoulder. Was continually bathed in exceedingly profuse perspiration. Was better the next day, and thought it was only a temporary illness. A week later was attacked with distinct chilliness, accompanied with vomiting and retching. On the eighth day feels better, but is very feeble. On the ninth day another and very severe attack, with dyspnœa and fainting; has pain in the epigastric region and in the sternum. Two days later another attack, with continued vomiting, exceedingly quick pulse, and complete prostration. A week later, after slightly recovering, she is again seized with a more violent attack than ever. In the meantime, distinct jaundice had developed. Dyspnœa and vomiting, followed by a complete collapse, and death the next day. Post-mortem, *soon after death:* The liver seems but little affected. The spleen (Figs. 1, 2) is more voluminous than usual, and resembles an oblong cone. Its tissues are dense. Its surface is smooth. The capsule (B) is similarly very dense. The whole organ is inclosed in a partly granular, partly fibrous pseudo-membraneous layer. The diaphragm, facing the organ, is equally red, and covered with fibroid exudate (D). The upper extremity of the organ has a mottled appearance of red and white. Fig. 2, a longitudinal section, showing the internal surface. The light-colored parts of the tissue correspond with the external fibroid deposits, and consist of great numbers of foci of solid pus (P P P). The darker parts (I S) are indurated structures, while (F P) consist of softened tissues and liquid pus. All other organs were normal.

FIGS. 3, 4.—*Softening of the Spleen.*

FIG. 3.—*Acute stage*, in pernicious paludal infection. There were thrombosis of the mesenteric veins and embolism of the superior mesenteric artery. Diffuse miliary hæmorrhages in the stomach, duodenum, and ileo-coecal region. The vessels of the liver were gorged with blood, but its tissues were pale and anæmic. The spleen (fig. 3) is of normal size, of a grayish color, and exceedingly soft, so that a slight pressure breaks the capsule and discharges a sanious, gray, semi-solid substance, leaving only the denser trabeculæ.

FIG. 4.—*Chronic stage of softening* in septicæmic hepatitis. The spleen pulpa is so soft as to be readily pressed out by a slight pressure of the finger. The organ is of a brown color. The capsule, also smooth, is without much consistence. The liver is infiltrated with bile, and is of an olive-green color. There was severe jaundice during life, and general septicæmic symptoms. All viscera are more or less affected.

likely to the various modes of investigation. Within the pulpous parenchyma there are always found great quantities of blood elements, which impart to it the peculiar reddish-brown color. The sacular enlargements on the arteries (the malpighian bodies) are true lymph organs, made up of adenoid tissues, and contain only lymphoid corpuscles. Their capillaries are extremely small and suddenly expand into wide veinlets. The lymph current in the spleen is contained in two sets of lymphatics—one extravascular, between the cellular and fibrillar structures, the other intravascular, that is, walled vessels, forming large plexuses on the outer walls of the arterioles and veinlets, and coalescing in wide networks on the hilus, anastomose with other lymphatics upon the mesentery. (TOMS, *splenic lymphatics.*) Great numbers of organic nerves accompany the vessels in the spleen and control its circulation.

The splenic function seems to be that of assisting in the formation of blood (both new elements and reorganization of the old); also in taking up and retaining a great number of foreign substances—both erogenous and exogenous—to the blood, a function proper to all lymphatic glands. (*Naunyn*, *Arch. d. Klin. Heilkunde*, *VI*, and *Berlin Klin.*) *Klein* (*Wochenschrift*, 1880) positively states that the spleen forms new blood corpuscles only in the fœtal state; with birth of the child this function ceases. On the other hand, *Foa* and *Salvioli* (in *Arch. per le Scienc. Medic.*), and *Bizzozzero* (in *Arch. Ital. Biolog.*) state that after hæmorrhages the spleen reproduces blood corpuscles even in adult life. *Tizzoni* (in *Atti della Accadem. real dei Lincei*, *Series* 3, *Vol. X*, etc.) states that after extirpation of the spleen in animals, there is regeneration of splenic structure in the shape of nodules, consisting of malpighian corpuscles, which contain nucleated young blood corpuscles. According to *Quinke* and *Gunkel* a portion of the iron of the blood corpuscles, when these break down, is deposited in the spleen, another portion passes into the liver, to be eliminated with the bile (as bilirubin?), and still another is used for organization of new blood corpuscles. Supernumerary or accessory spleens are often found attached to the splenic vessels. They differ in size from that of a small pea to a very large bean. They are usually situated near the hilus of the organ, and are made up of regular splenic tissue. During digestion the spleen swells up, but afterward becomes rather contracted.

In all infectious and contagious diseases the spleen becomes affected with congestion and hyperæmia. In the early stages of typhoid fevers, acute eruptive diseases, syphilis, pyæmia, etc., the spleen swells often enormously; all its vessels and its parenchymatous tissues are gorged with blood and the capsule tensely distended. The pulpa structure is of an intense red color and very soft. The malpighian bodies are then either enlarged and plainly visible, or are covered by the pulpa structure. This congestion may be of short duration or persist for some time, and gradually undergo morbid changes. In a state of chronic congestion the organ has a grayish-red or gray color, usually much enlarged; it sometimes becomes of enormous size. The gray color is due to infiltration with great quantities of lymphoid cells, derived either from the general circulation or from the organ itself. The capsule is then opaque, and on its surface it is covered with quantities of fibroid pseudo-membranes. Many cells in the pulpa become enlarged and nucleated, or have a vesicular appearance. Some contain blood corpuscles or their detritus. Many foreign substances and much infectious material are then found therein. (*Birch-Hirschfeld*, *Friedreich*, *Socoloff*, *Fischl.*) After the inflammation subsides the organ returns to its normal size, but traces of the hyperæmia often remain in the shape of pigmentation of the tissue. This most frequently happens after repeated acute attacks, as in malaria, etc. Upon the capsule raised places of different shapes also remain behind, and occasionally these hyperplasiæ give rise to adhesions of the organ to the adjacent tissues or organs. The size of the organ undergoes many changes by inflammation and subsequent anæmia. The volume may become extraordinary large, filling the greater part of the abdominal cavity, and its weight may increase to eight or more pounds. After many repeated attacks of intermittent the size steadily increases, and ascetis is often caused by its pressing upon the veins of the abdominal viscera. Atrophy or indurative processes reduce the size of the organ to an almost insignificant size. When the pulpa contains little or no blood pigment its color is a bright red. The greater the quantity of pigment the darker will it appear. It will become dense, often very hard, the vessels become indurated and stained with brown, yellow, or gray pigment; the same will be the case with the trabeculæ and other reticular structures. The coloring matter is derived from great quantities of destroyed blood corpuscles.

Acute splenitis very seldom leads to suppuration. It is only when infectious matter containing bacteria accumulates in the organ in great quantities that suppuration takes place, or abscess is formed by extraordinary quantities of exuded spheroid cells in the pulpa, which compress it and produce pernicious anæmia in the organ. The result is then either diffuse suppuration (which gives the pulpa tissue a yellowish-white appearance) or localized abscesses with subsequent gangrene. Local suppurations are more frequent than diffuse, and are also less dangerous. *Ponfick* found abscesses in the spleen to be frequent in pyæmic infection and typhus recurrens. While the abscess is grayish yellow or gray, the adjacent tissue becomes discolored (pale white or deep gray) and infiltrated with serum and pus. It frequently happens that the abscess perforates its capsule and peritoneal covering, and discharges its contents into the peritoneal spaces; this invariably leads to death. When granulation is formed upon the suppurating surface, it is liable to cause adhesion of the organ to its adjacent structures. This condition is often followed by many complications (*Besmer*, *Ponfick*). Obstruction of the splenic vein is always followed by congestion. In many diseases of the liver, the lungs, or the heart, such obstruction in the splenic vein frequently takes place. Hepatic cirrhosis is the most frequent cause of splenic congestion. In this disease the portal branches in the liver are obliterated, the venous blood from the splenic vein can not readily be removed, and the splenic tissue becomes congested, then indurated. The color of the pulpa is either bright or dark red, according to the quantity of newly-formed connective tissue in the organ. In splenic anæmia, subsequent to very profuse hæmorrhages, the tissue turns pale and is very lax.

Embolic infarcts are sometimes produced by detached particles from vegetations on the inner surface of the heart or the aorta, which are usually followed by gangrene of the anæmic tissue. When the plug becomes absorbed, that is, when the infarct is not extensive, or the embolus has no infectious properties, very fine granulations are formed, with subsequent formation of dense, fibrous, cicatricial connective tissue and contraction. Infectious emboli generally lead to pyæmic infection, suppurative splenitis, and sphacelus of the tissue, usually ending in the death of the affected person.

General atrophic conditions of the body superinduce atrophy of the spleen. In old age splenic atrophy is the rule. All its tissues suffer from this atrophy.

Amyloid degeneration of the spleen is found in two forms, the granular and the lardaceous. In the granular (*sago-spleen*) the malpighian follicles are affected. They appear as light-brown, translucent granules within the dark-red structure of the pulpa, are larger than normal, and resemble boiled granules of *sago*. Treated with iodine dissolved in a solution of iodide of potassium they turn a *mahogany* color. The lardaceous form is only a diffuse degeneration of the tissue. The organ becomes thus much enlarged, and a cut surface presents a hyaline appearance. This morbid alteration primarily affects the vascular walls and the trabeculæ; the pulpa cells and the follicular structures undergo only secondary alteration from the pressure exerted upon them by the enlarged morbid tissue (*Virchow*, *Kyber*, *Eberth*).

Spontaneous rupture of the spleen from enormous swelling, or after wounds, sometimes takes place, and usually proves fatal from the enormous hæmorrhage accompanying it. If a thrombus is formed in the broken vessel and the bleeding stayed, recovery is possible.

In general tuberculosis of the body, tubercular eruptions in the spleen are always met with. The eruptions are of miliary size in miliary tuberculosis of the body. In chronic tuberculosis various-sized caseous *nodes* are formed in the malpighian bodies and in the walls of the arteries and their sheaths. Gummatous formations are not frequent in the spleen; still, in hereditary and even acquired syphilis, gray, translucent nodules are found in the pulpous structure. They sometimes appear as opaque, yellowish-gray tubercles. In hereditary syphilis of children the spleen is very much enlarged, its arterial sheaths are diffusely infiltrated, and the stroma augmented.

TABLE VIII.

FIGS. 1, 2, 2*a*.—*Acute Splenitis, with hæmorrhagic infarcts and abscesses in the splenic tissue.* In both cases there was septicæmic infection, abscesses on the liver and in the lungs. In both, also, all the organs of the body contained pus. The primary lesions were traumatic hepatitis, wounds received in a street fight. In both cases the spleens were enlarged, and the capsules (*a*) very thick. In some portions the pus was liquid in many, in the form of a semi-solid puriform focus. The vessels were not much altered, but the trabeculæ in the pulpa were visibly enlarged, and very readily perceptible as thick red strings or fibers. A great number of hæmorrhagic points were observable on the capsular surfaces (*a*) and in the pulpa.

FIG. 3.—*Splenitis in Pulmonary Tuberculosis.* The patient a child twelve years of age. In the lungs, the bronchial and mesenteric glands, there were great quantities of tubercular deposits in all stages of metamorphosis. In the glands at the splenic hilus, which were much enlarged, a great quantity of miliary and larger tubercles were observed (G T). In the spleen the tubercles were disseminated in all the tissues. The pulpa was of a deep red. Within the malpighian bodies they had a lighter color.

FIGS. 4, 4*a*.—Spleen of a four-year-old child who died of acute miliary tuberculosis. Vast numbers of miliary eruptions existed in the liver (in the interlobular structures). They were white, transparent nodules, singly or in groups. In Fig. 4*a* they were in an advanced caseous state. In the spleen (T T), both in the capsule and in the pulpa, they were alike disseminated. Portion of the peritoneal covering is left to show the eruption on the capsular surface. Fig. 4*a*, a longitudinal section of the organ, shows that the pulpa is as much affected as the capsule.

(*Barensprung, Hereditary Syphilis; Wagner, Arch. d. Heilk. IV; Mosler, Klin. Wochenschr.*, 1864; *Gerhardt, Lehrbuch; Birch-Hirschfeld, Arch. d. Heilkunde*, 1875.)

There is a form of splenic enlargement the cause of which is as yet not definitely determined, but which forms a most important symptom of a disease which often proves fatal, that is, *leucocythæmia.*

The relation of this hyperplasia to leucocythæmia is still a subject of difference of opinion among high medical authorities. *Virchow* and *Bennett*, having found that the existence of splenic hypertrophy was nearly constant in leucocythæmia, have concluded that the hypertrophy or hyperplasia, as the case may be, was due to enormous increase of leucocyths in splenic tissue by an exaggerated function of cell production in that organ itself, or in the lymphatic glands, and their great abundance in the blood, to their passage through the splenic vein. *Griesinger* thought that the hypertrophy was only the consequence of accumulation of leucocyths in the organ, but not due to over-production therein. In 1832 *Hodkin* published a number of cases of general leucæmic hypertrophy of all the lymph-glands in the body. Since then a number of similar cases have been reported. In 1874 *Demange* described similar cases, which he designated as lymphadenitis, but in which there was no general leucocythæmia. Most of the French clinicists consider leucæmia not as a primary and separate disease, but as a secondary cachexia following many pyæmic and septic diseases. There are cases of lymphadenitis and splenic hypertrophy without leucæmia, and *vice versa.*

The liver is one of the organs oftenest affected in this lesion. This was already noticed by *Bennett* in his thesis, "Hypertrophy of the Spleen and Liver." The structural changes taking place in the liver in leucæmia vary according as the leucocyths affect the vessels or form focal deposits. In the first case there is enormous influx of those corpuscles in all the capillaries of the liver until they dilate very much in some places, and, driving out the blood from the cavities, still further distend the vessels, thus compressing the lobular cells, the interlobular vessels, and the biliary ducts and capillaries. Hæmorrhages and infiltrations, with subsequent sclerotic hypertrophy of the perivascular structures, then follow. In the second form there are formed in many places, on the surface and in the parenchyma, a great number of nodules, varying in size from that of a mustard seed to that of a large pea. They are of a gray color and are rather dense. They stand in no connection with the vessels (*Robin, Isambert, Memoires; Olivier* and *Ranvier, Observations*, 1867).

The functional disturbance produced in the liver by the vast quantities of leucocyths can readily be conceived from the radical changes in the tissues of the organ. The whole portal circulation becomes gradually obliterated, and ascites from dilation of the abdominal sub-cutaneous veins are produced. Neither glycogen nor bile can be produced to any extent, if at all. The whole body undergoes a state of cachexia. *Leudet*, in *Gazette Medic.*, 1858, and *Hayden*, in *Dublin Quarterly Journal*, 1865, describe cirrhotic conditions of the liver found in leucocythæmia. *Virchow*, under the name of *lymphoma*, describes a number of cases in which these lymphadenic structures were found to exist in many places in the body, especially in the liver of leucæmic patients. They were in such abundance as to give the organ a mottled appearance. He considers them to be the product of cell proliferation within the hepatic and other parenchymatous connective tissue. As the liver thus becomes filled with leucocyths, they pass into the bodily circulation by the hepatic vein and produce leucæmia.

As the portal-vein circulation becomes more and more obstructed there is formed a stagnant condition of the splenic circulation; the spleen retains a vast number of leucocyths, and thus becomes secondarily affected with hyperplasia.

Even under ordinary circumstances the blood in the splenic vein contains three times the number of leucocyths contained in other blood. This well-established fact has given rise to the theory of blood-formation in the spleen, as it was assumed that those cells were young blood corpuscles. No direct proof of it has, however, been furnished until now. This theory has led to another, that is: the overproduction of leucocyths by the spleen causes the hypertrophy of the organ in leucocythæmia. One fact has been overlooked by those holding that theory: that total absence of the spleen after its removal from the body does not at all change the ratio of the colorless to the colored blood corpuscles.

Virchow describes splenic hyperplasia (in *Gesammelter Abhandl*, 1856) as follows: "A cut surface of the tissue is often found perfectly anæmic, or of a more or less red color, pale or yellow. It is moist, dry, and uniform. Only the larger vessels seem to be open and dilated more than usual; the follicles are smaller than normal, often nearly invisible, of a paler color than the pulpa. This latter is highly developed, elastic, and very resistant. The trabeculæ form white bands, and contrast with the color of the capsule. Also, the elements are normal, yet there are more of them. Yellow, red, gray, or black pigment are in plenty. The tissue is indurated. He considers this similar to the state of the spleen found in paludal hypertrophy. *Tarchanoff* and *Swaen* (*Acad. de Scienc.*, 1875, and *Kelsch, Arch. de Physiol.*, 1875), throw much light on the sclerotic alteration of the spleen in paludal infection. By severing all the nerves entering the spleen, *Tarchanoff* and *Swaen* found the organ very much tumefied, and noticed that it retained great quantities of colorless corpuscles, which diminished in quantity in the general circulation. A similar condition of the spleen was found by *Kelsch* in paludal fever. During the fever paroxysms the spleen swelled up, but contracted after the paroxysms. *Botkin* (*Milz contraction*) found that when contraction of the enlarged spleen, in fever, is produced by electricity that it diminished in size, and the liver increased in volume, and great numbers of leucocyths are then found in the last organ. In the later stages of paludal cachexia, the fibrous structure in the spleen predominates, the follicles become atrophied, and connective-tissue bands substitute the lymphatic elements. Similar phenomena exist in leucocythæmic hypertrophy. In the latter the enormous accumulation of those cells is permanent. The irritation they produce upon the tissues calls forth inflammatory processes, after which secondary parenchymatous changes take place. Very likely a notable quantity of cells enter through the splenic vein into the liver, and eventually into the blood.

According to *Chas. Robin* (*Journal de la Physiologie*, 1859), the leucocyths, like all other anatomical elements, are produced by cell-division, under various circumstances, at the expense of some proximate principles of the blood plasma or lymph. According to *Ranvier* leucocythæmia constitutes the last and fatal stage of many septic and pyæmic infections. Pouchet, Sepalier, Potain, Lagrave found a leucæmic state in very numerous cachectic diseases. Renault in a very able memoir on the subject published in 1881 states: "I have observed that in many lymph globules the nuclei were in a state of fission. Considering the different sizes of the leucocyths in leucæmia, one can not help drawing the conclusion that they originated and grew in the blood itself. The nuclei in some cells are surrounded by more protoplasm than in others, presenting characteristic cell-growth. * * We were enabled," he continues to observe, "that colorless corpuscles, which were undergoing fatty or granular changes, were in a state of segmentation. A shining globule of an almost spherical shape suddenly contracts in the middle, assumes the form of a dumb-bell, and in a short time is divided into two cells." (*G. Variot*, in *Journal de l'Anatomie*, 1882.)

In all acute fevers and in most cases of extensive inflammation of the parenchymatous organs, the spleen is found, on close examination, to be changed in form and consistence, usually more or less enlarged. The lighter forms of alteration in the tissues of the spleen consist of massive infiltration of lymphoid bodies into the pulpa, which speedily disappear after the inflammatory processes in the body cease. In the severer forms the spleen becomes hyperplastic, the pulpa tissues as well as the malpighian bodies are in a state of true hypertrophy, and the enlargement produced does not disappear with the inflammations in the body. In infectious diseases the organ attains sometimes an extraordinary size. This increase in volume is due to a peculiarity of the structural arrangement and the vascularity of the spleen. Its circulation is always slow; the meshes are always full of detritus of the blood and of all kinds of useless and foreign debris of substances introduced in the blood. In infectious fevers there is enormous accumulation of corpuscular virus in the organ, which reacts against the irritation by hyperplastic inflammation. The enlargement is generally the first and often the only symptom of the infection in its early stage, as it is very often the only one remaining after all others have disappeared. It is in this respect very much like the enlargement of the lymphatic glands, an early and a late symptom. In the early stages it will form the prodromal indication to warn the patient of the coming danger, as in the latest stages it will show that the danger is not yet over and a renewed attack of the fever may be expected. The accumulation and retention of infectious substances in the spleen often give rise to formation of pus and abscesses, which is then manifested by acute pain in the splenic region. Yet such conditions seldom exist in typhoid, intermittent, or remittent fevers. They are mostly found in typhus recurrens; in variola, especially the hæmorrhagic form, and in acute tuberculosis. The pain produced in the spleen is communicated to the left shoulder, the arm, and often the whole left thorax, simulating pleuritic pains. As a rule, such pains are coincident with extreme pain in the joints, and resemble, to some extent, arthritic rheumatism, from which it differs by an absence of all swelling in the joints and of pericardial symptoms. The pains in the joints are due to infiltration with infectious matter and pus, or bacteria in the epiphysal portions of the bones. In osteo-myelitis the pain in the spleen is sometimes extreme, in fact, severer than in the affected joint. It often appears before any swelling of the articular surface of the inflamed bone, and misleads the patient and physician to suppose the existence of some form of miasmatic fever. When the fundus of the eye is examined at this time it is found that a number of whitish spots are disseminated over the retinal surface, with very severe congestion or turgescence of the veins near the papilla.

ACUTE HÆMORRHAGIC SPLENITIS.

SPLEEN IN PULMONARY TUBERCULOSIS.

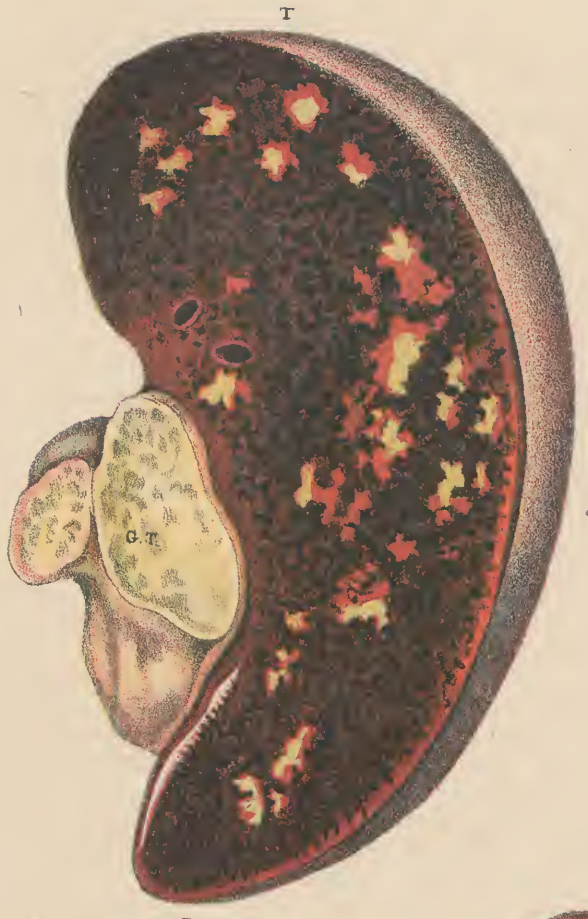

Fig. 3.

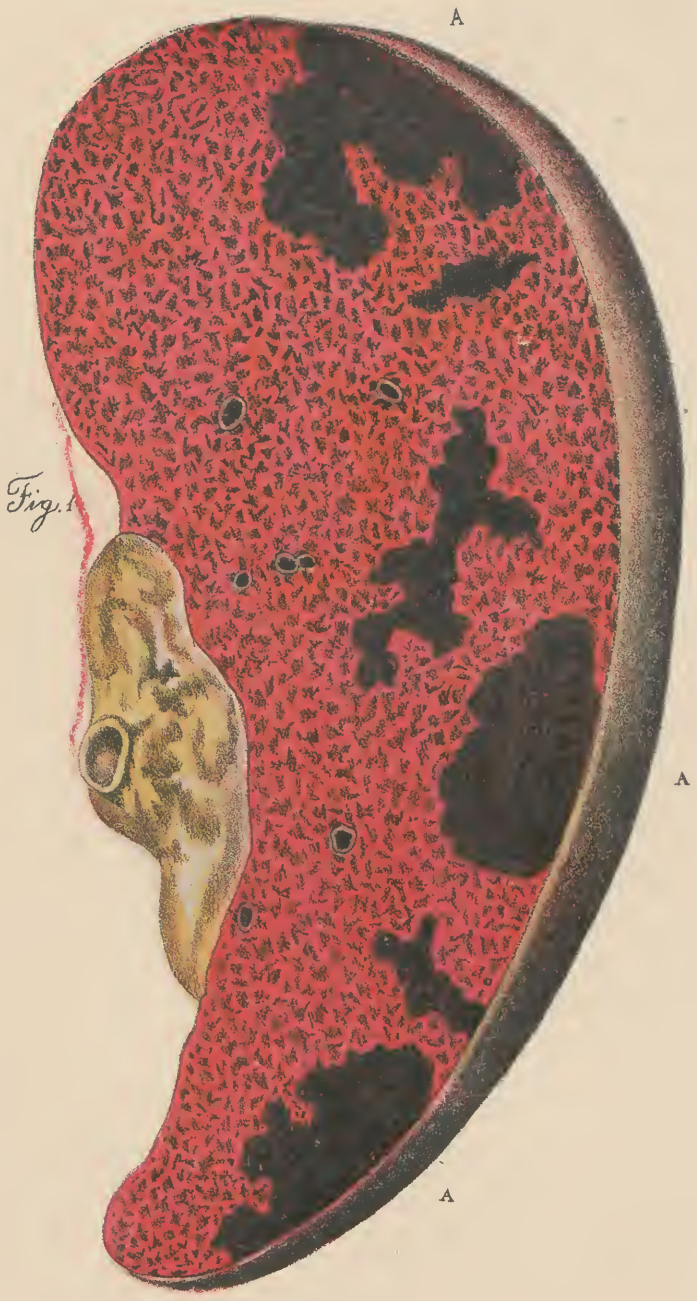

Fig. 1

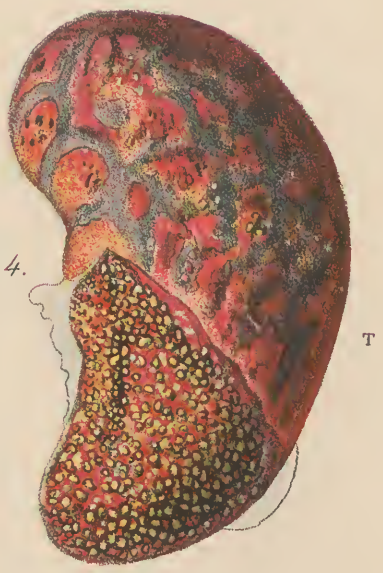

Fig. 4.

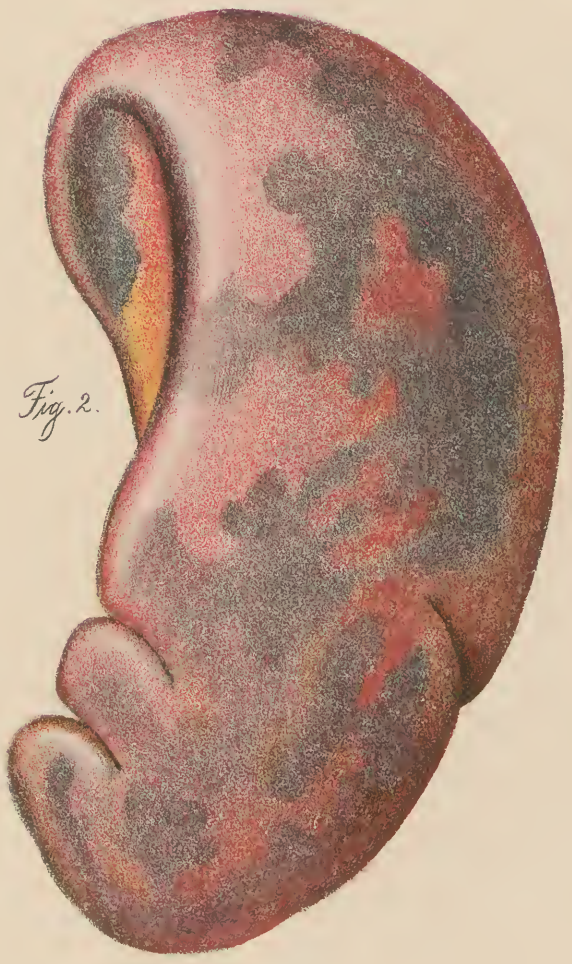

Fig. 2.

Fig. 4a.

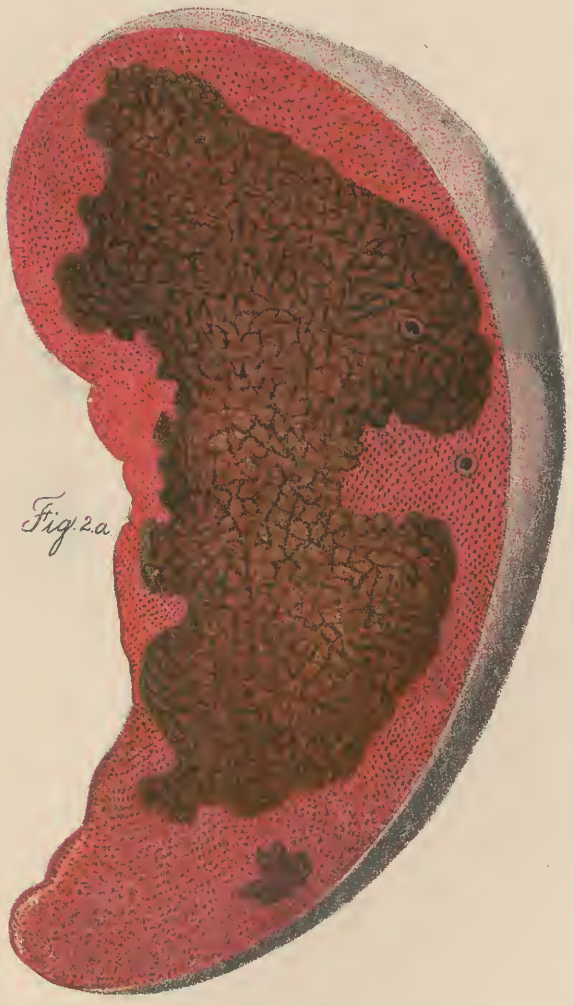

Fig. 2a

DISEASES OF THE URINARY APPARATUS.

Sec. VIII. *Tab. I.*

CHRONIC INDURATIVE NEPHRITIS.

AMYLOID CHANCE OF THE KIDNEYS.

Fig. 1.

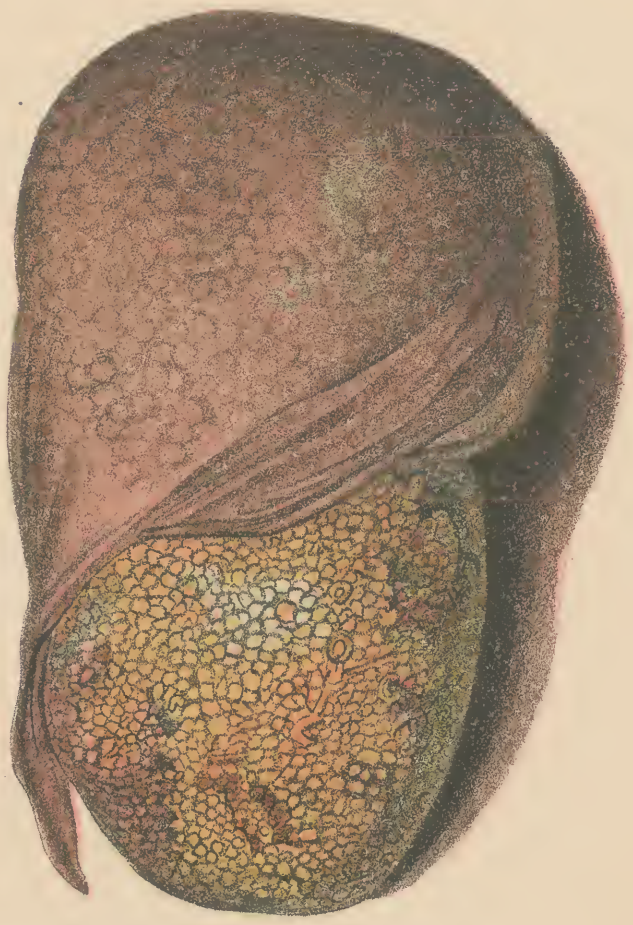

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

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TABLE I.

FIGS. 1, 2.—*Chronic Nephritis and Induration.*

CASE.—J. K., thirty-four years old (a patient in Guy's Hospital, London), under the care of *R. Bright* (*Report of Medical Cases*). The patient had been a sailor, but had given up his business. Was in the habit of taking considerable quantities of spirits; had lately taken none. About six days before admission into the hospital he was seized with pain in his loins, knees, and ankles; his legs soon became much swollen, and his hands and face œdematous. After admission: the abdomen was painful on pressure; pulse 78, rather hard; tongue natural, but pale; bowels slightly purged; urine scanty; appetite good. Stayed in the hospital six weeks, alternately improving and relapsing. Shortly before death permanent anasarca set in, with painful dyspnœa, and symptoms of heart trouble manifested. Died in about seven weeks after admission.

Autopsy: Countenance bloated, of purplish color; legs œdematous. The pericardium contained about four and a half ounces of clear serum. Many patches of villous deposit of fibrin of recent formation within the pericardium. The heart is enlarged and firm. In the angle between the aortic semi-lunar valves a solid ossoid structure was formed. The left lung adherent throughout, and the whole tissue in a state of slate-colored induration. The right lung soft and œdematous. A moderate quantity of serum within the whole thoracic cavity. Very copious effusion in abdominal cavity. Some alteration in the mesenteric glands. The liver is nearly normal. Spleen dark, and covered with fibroid pseudo-membranes. The kidneys granular throughout (Fig. 1, this section). The surface is rough externally, all trace of natural organization obliterated, with the exception of the pyramids, which were of a lighter pink color than usual. Fig. 2, longitudinal section of the same kidney.

FIG. 3.—*Amyloid Change of the Kidney Tissue in Hæmorrhagic Nephritis.* M. S., a woman of twenty-five years; has led a very irregular life, and was at one time much given to strong drink. Present symptoms: Very frequent respiration, cough, and dyspnœa. Symptoms of hydrothorax. The face bloated and of livid color; legs œdematous; involuntary discharge of urine and stools; albumen in urine. Died of suffocation. Autopsy: Considerable quantity of serum in thoracic cavity. Upper part of left lung œdematous, and tissue dense. Tubercles in early stage. Pneumothorax in right thoracic cavity. Right lung dense and infiltrated. Apex of same lung filled with tubercles; two suppurating cavities in same. The heart nearly normal; origin of aorta atheromatous. Serum in abdominal cavity. Pale yellow liver. Ulceration in ileo-cœcal region of intestines. Agminated glands ulcerated. Kidneys disfigured by nodular surface. After removal of the tunic the surface of the organ presented numerous white nodules upon a pink ground. Stellated veins in several places. Size of organ but little changed. Proper tunic closely adherent. Internally the whole structure of the cortex of a nearly uniform yellowish drab, with small opaque yellow spots. Many vessels were obliterated.

FIGS. 4, 5.—*Portions of Kidney of the same person.*

FIG. 6.—*Amyloid Degeneration and Induration.* The parenchyma is spotted. The cortical portion indurated, shows no remnant of the tubular structures; the outer surface is congested, and in parts shrunk.

Anatomy of the Urinary Organs.

The urinary apparatus consists of the kidneys, the renal pelvis and ureters, the urinary bladder, and the urethra. Of these the kidneys alone are, strictly speaking, excretory organs; the others are either collecting or drain tubes, to carry the urine from the body. The structural arrangement of the kidneys is such that the blood passing through their arteries loses, by means of a peculiarly-adjusted system of tubing, much of its water, many of its saline and other inorganic and organic substances unfit for circulation in the body, and retains its albuminates.

Anatomically the kidney consists of the capsule, a tough, fibrous membrane inclosing it, the organ itself made up of the pelvis, the collecting cavity, a dilated portion of the ureter, and the parenchymatous glandular structures, blood vessels, nerves, and a few lymphatics. The glandular structure proper consists of a complex system of tubing, which goes to make up the two functionally-different parts, the cortical and the medullary, with their respective specially-arranged vascular distributions. The medullary substance is denser and paler than the cortical, and is marked by a radiary, striated appearance. The striæ are arranged in cones, their bases facing outward, the apices turned toward the hilus, constituting the pyramids. Each apex discharges, through its perforated surface, the urine into the renal pelvis. The cortical portion contains more blood than the medullary, and hence usually looks darker than the other; it is softer, and has no striæ. It envelops the pyramids, except at the apex, and constitutes the greatest bulk of the kidney tissue, occupying not only the whole exterior portion of the organ, but also filling the spaces between the pyramids, forming the so-called columns of BERTINI. These columns are made up of cortical coverings of the pyramids, between which a quantity of connective tissue, containing the vessels and nerves of the kidney, is lodged. Each pyramid, with its cortical envelope and vascular network, constitutes a lobule of a multiplex tubular gland.

In man the lobules are so close to each other as to appear as one single organ. The cortical, as well as the medullary, substance consists essentially of blood vessels and smaller or larger tubules—*the uriniferous tubules.* They begin in the cortical part as spherical dilatations or capsules, inclosing internally a little ball of vascular loops (the glomeruli) and soon contracting form a sort of a neck to the capsule called MALPIGHIAN, and after assuming a tortuous course—at the same time becoming somewhat wider than at the neck—in a serpentine, zigzag fashion, pass toward the apex of the pyramid in the form of a loop, where they contract and pass between the branches of the straight tubules, then bend backward again, widen a little in their passage back into the cortical part, and there form a junction with or a branch of a straight tubule. The loops are called those of HENLE. The wider serpentine tubules are called *tubuli contorti.* The straight tubules, which receive the ends of the tortuous loops, pass into the medullary portion, or rather form the central part of the apex of the medullary cones, where they form the wider trunks by the union of the many straight branches of those running from the cortical into the medullary portion. In and a little above the apex of the pyramids these trunks are formed into short dilated stems, opening into the pelvic cavity (*Ludwig*). Most of the peripherally ascending tubules are the ones which form the tortuous canals and carry on the excretory functions, whilst the central are merely conducting tubes, which receive and carry the urine into the renal pelvis. The urinary tubules consist of a proper membrane, internally invested with epithelium. The adventitia is of homogeneous structure. The one-layered epithelium has a different character in the several parts of the course of the tubules. In the tortuous portion the cells are not clearly delimited; the nuclei are imbedded in a softish opaque mass; the protoplasm is granular and usually contains fat molecules. In narrower portions and the tubuli recti the cells are simply cylindrical. The papillary ducts have no limiting membrane (*Ludwig*). In the malpighian capsules are well defined cells; they invest the glomerulus all over like a fine sheath.

The branches of the renal artery divide in the pelvis into secondary vessels, which enter into the columns of BERTINI, and here form a network around each pyramid. From these embranchments, which reach the cortical boundary, a number of branchlets regularly ascend at right angles into the cortex and here give off branchlets also at right angles to their own axes and ascend between the contorted tubular bundles in a somewhat tortuous manner to the very surface of the kidney, where a number of their superficial branchlets anastomose with the capsular branchlets. *Koelliker* called the ascending arteries interlobular. Their rectangular branchlets form the glomeruli, like berries at the tip of fruit-bearing twiglets. The horizontal branchlets are many, and form many malpighian capsules with the widened portions of the contorted tubules. The capsule consists of afferent arterial branchlets, the vascular loops, and an efferent branch, which still retains its arterial characteristic structure, but which subdivides into a vast network of very fine capillaries, which spin all over the tortuous canaliculi a vast network of rectangular meshes.

The *vasa afferentia* bounding on the medullary portion are wider than the above-described, and pass into the midst of the *tubuli recti* and here form the *arterioli recti*, where they divide and form straight branchlets like the straight tubules, giving off branches which form capillary networks in the pyramids. At the boundary between the cortical and medullary parts the capillary meshes of contorted tubules inosculate with those of the *arterioli recti.* A portion of the straight arterioles also are derived from the branches of the interlobular arteries. On the surface of the kidney the veins form stellated trunks from the veinlets of the interlobular branchlets. They coalesce and enter the interlobular spaces by the side of the arteries and take up with the smaller veinlets from the interior of the cortical substance, inosculate with similarly-running veins, forming secondary arches, and reach the pelvic trunks by the side of the arteries. They are all devoid of valves. Before they leave the kidney they receive the papillary veins, which form circular meshes around the orifices of the tubular trunks of the papillæ. The lymphatics are few. They accompany the arteries in their course.

Pathological Anatomy and Pathology of the Kidneys.

The morbid changes of the kidneys may be divided into several groups: 1. Those produced by disturbance of renal circulation. 2. Those brought about by infiltrations and exudations of corpuscular elements into the tissues from the blood, or accumulated by cell division of the tissues themselves (*hyperplasia*, *heteroplasia*). 3. Changes brought about by inflammatory degenerative processes, superinduced by alterations of the constituents of the blood, and by its impurities. 4. Tumors, hyperplastic and heteroplastic formations.

Hypertrophy and Atrophy—Primary Diseases of the Kidneys.

Hyperæmia is either the result of increased aortic pressure, or depression of the renal arteries. Excretion of urine chiefly depends on the velocity and pressure of blood in the glomeruli. Hyperæmia consequently increases the elimination of that liquid. Destruction of one kidney produces vicarious over-activity of the other by an increased quantity and pressure of blood therein. Under such circumstances the remaining kidney may increase in size and become hypertrophic. When a portion of a kidney is destroyed the remainder is supplied with a greater quantity of blood for the performance of its function; it may then also become hypertrophied. In the enlarged and dilated tubules the glandular cells are increased in size and number (*Leichtenstern*, *Beumer*, *Perls*, *Eppinger*, *Ribbert*, *Grawitz*, and *Israel*). Static hyperæmia is due more to general than to local disturbance of circulation. It may take place in diseases of the lungs or the heart, or in local troubles, such as thrombosis of the vena cava and the renal vein. In the first weeks after birth babes are often thus affected.

When renal venous circulation is suddenly checked the kidneys become engorged with blood, swell up, and assume a purplish or brownish hue. Hæmorrhages speedily take place, both in the capsules and in the medullary structures; the tubules become gorged with blood. When venous circulation is slowly impeded, partial circulation is re-established by collateral venous circulation through the small veins which enter the renal capsule and anastomose with other small veins of the phrenic, lumbar, and supra-renal regions. When stagnation of the venous current persists for some time the kidney tissue undergoes fatty degeneracy and decay. When the obstruction is not extensive the kidney swells up and has a purplish

TABLE II.

FIGS. 1, 2.—*Chronic Diffuse Nephritis (Granular Kidney of Bright).*

CASE.—C. S., aged forty. Low-conditioned and dissipated woman (at Guy's Hospital, London). Has been repeatedly treated for dropsical trouble. Attributes her present attack to exposure to wet and cold. Present symptoms: The whole body anasarcous. She passes but little water. Breathing much oppressed. Urine albuminous. She states that for the last six months she had pain in the loins. At present she has a sense of weakness and pressure there. The thoracic symptoms become steadily more and more aggravated, the quantity of urine diminished; the greatest portion of it is albumen. She is gradually sinking; coughs, and expectorates much blood. Dies of exhaustion.

Necropsy: Much serum in abdomen. Liver slightly lobulated; acute margin rounded; the organ enlarged; acini light-colored; interlobular structure bright red. Some bile in gall-bladder. Kidneys small, rather lobulated, of semi-cartilaginous consistence, completely granular. Small white and yellow projections above external surface, which is red and looks rough. A longitudinal section presents a hard surface; the pyramids drawn outward; the cortical portion slightly granular, and of a drab color mixed with purple spots. In Fig. 1 the tunic is removed to show the contracted granular external surface. Fig. 2, longitudinal section of the other kidney of the same person. The granular texture of the whole cortical portion and the peculiar manner in which the pyramids are drawn outward indicate the infiltrated condition of the parenchyma.

FIG. 3.—*Recent Glomeral Nephritis.* A longitudinal section of a part of a kidney in a state of recent inflammation. Softening and obliteration of the pyramids. The whole cortical part is of a pale color, and interspersed with small yellowish opaque spots. There were albuminuria and extensive dropsy in the case.

FIG. 4.—*Recent Glomerulo-Nephritis, with Thrombosis of the Renal Veins.* The kidney was enlarged, very dark on its upper surface, and on the lower mottled yellow. On external surface, yellow raised granules. Internally, the substance remarkably pale, with a slightly-granular lardaceous appearance. There were very severe pleuritis and peritonitis, attended with weak heart and low blood pressure. The recent hydrothorax and extensive œdema showed obstruction of the venous circulation in the body, especially in the abdominal and thoracic cavity. Many veins were filled with thrombi.

FIGS. 5, 6, 7.—*Large White Kidney.* The case is of a dissipated and drunken man. His health was generally bad. Was taken shortly before death with swelling of the whole body and consecutive anasarca. The apex of the left lung was found in a state of tuberculosis. The pulmonary tissue was soft and œdematous. The kidneys were white, somewhat enlarged, and lobulated; very soft, and not granular. Some stellated vessels on external surface. The whole cortical portion was found on a longitudinal section to be uniformly whitish-gray, without any trace of the normal structure. The pyramids were faintly colored. Fig. 7, a portion of the kidney macerated for some time to show the granular texture of the surface.

color, is *cyanotic*. When the kidney persists in this state for some time it becomes indurated and solid. The cortical portion is pale or grayish red, and along the tracks of the veins a little striped. This state is named cyanotic induration. When venous stasis exists only a short time the vessels of the kidney become gorged with blood, the smaller veins and the capillaries considerably dilated, within the malpighian capsule and in the uriniferous tubules there is found some liquid, which forms a precipitate by boiling, and some blood corpuscles. Within some of the tubules hæmoglobin and casts of the tubular cavity are found. These are the so-called albuminous or fibroid cylinders. They are made up of albuminates, which have passed with the watery constituents of the blood, from the glomeruli, and have become coagulated in the tubules. The epithelial cells of the looped tubules contain yellow and brown, partly crystalline pigmentary substances, derived from the partly-destroyed extravasated blood corpuscles. When stagnation has existed for some length of time and the kidney has become indurated there is formed an increased quantity of interstitial connective tissue. The blood vessels become dilated, their walls thickened. The tubular epithelium undergoes fatty change, especially in the straight tubules of the cortical portion. The glomeruli are usually not much altered; however, here and there a malpighian corpuscle becomes changed into a solid spherical mass and atrophied, and the capsule and the adjacent tubule collapse and contract.

Secretion of urine, under such circumstances, is very much diminished, and the little that is evacuated contains blood corpuscles and albumen which, according to *Conheim* and *Senator*, are derived from the capillaries surrounding the malpighian apparatus. This albuminuria is less due to disturbance of circulation of the renal artery than to morbid change in the secreting membrane of the glomerular epithelium. (*Litten and Buchwald, Vir. Arch. Vol. LXVI, Hortoles, Etude du processus, Paris*, 1881, *Traube, Gesammte Abhandlung, Weissenberger, Arch. f. experim. pathol. Vol. VI, Posner, Virch. Arch. Vol.* 79, *Heidenhein, Herrmann's Lehrbuch.*)

In general anæmia, in contraction of the renal artery or its branches, the kidney becomes anæmic. If the anæmia is very considerable the organ has a pale, grayish-white appearance, somewhat translucent. When the anæmia is not general it is spotted red and white. The secretion of urine is diminished in measure as there is a lack of blood in the organ. When the anæmia is general there will be albuminuria. Such conditions exist in cholera, in epilepsy, tetanus, lead colic, and similar diseases. According to *Conheim*, the presence of the albumen in the urine is due to a degenerated state of the glomerular epithelium, caused by want of circulation. In anæmia of short duration there is hardly any trace left behind, post mortem. When the anæmic state exists a long period the changes in the glandular tissue and glomeruli are very manifest. Insufficient supply of oxygen to the kidney tissues produces a fatty change, and great quantities of fatty masses of different sizes give the organ a speckled appearance of gray and white. Total suppression of renal circulation produces gangrene of its tissues. When partial anæmia exists a long time in the kidney there are produced marasmus and shrinking of the structures, the epithelium decays, the glomeruli and the smaller vessels are morbidly altered, the whole malpighian capsules shrink and are changed into solid spheroid masses, the capsular membranes collapse, the vascular loops are obstructed by thrombi, the vascular epithelium becomes obliterated. The glomeruli thus affected lose their transparency, their afferent portion is either closed up or directly communicates with the efferent vessels; the tubule connected with the capsule also undergoes atrophic change; the epithelial lining loses its characteristic form and is changed into ordinary non-functional epithelium. The cavity of such tubules becomes filled with cylindrical or globular, fibroid masses, some of which distend the tubules into cystoid enlargement, and the masses assume sizes readily visible to the unaided eye. The fibroid masses are derived from the albumen of the decomposed cells and remnants of secreted albumen. There is no increased interstitial connective tissue, but there may be cellular infiltration in the existing tissue. *Overbeck* and *Herrmann* have shown that interruption of renal circulation for a short time will give rise to albuminuria, which will persist for a time even after the circulation has been re-established.

In old age atrophic conditions of the kidneys take place, and are recognized by shrinking of its surface and a more intense red color than the balance of the organ. Various atrophic conditions also exist in many diseases of the kidneys, such as embolic closure of the renal artery in the several forms of nephritis and hydro-nephrosis, and especially in arterio-sclerosis. This morbid alteration is very common in old age. It consists in a thickened and condensed state of the arterial walls. When the inner coat of the artery is considerably thickened, and its cavity is thereby more or less constricted, there is formed destruction of the tissues and obliterations of the glomeruli, the larger the size of the vessel and the greater the region it has to supply. Obstruction and obliteration of one afferent branch implicates only one glomerulus, but when a whole interlobular artery is thus affected a whole group of these becomes involved. Usually the lesion appears in spots in several regions of arterial supply. There are cases in which the cortical portion is uniformly affected in this manner. Confined to isolated spots these have a contracted appearance. The surrounding tissue looks grayish red, the affected spot dark red. The more numerous the affected localities the more cicatricial contractions on the kidney surface. It assumes, in intense cases, a granular appearance, the surface looks embossed, the whole volume of the organ is much reduced, sometimes to an excessive degree. In such an affected kidney the glomeruli are nearly all obliterated, the uriniferous tubules atrophied, collapsed, either empty or filled with altered epithelium. Many tubular cavities, especially those of the loops of HENLE, are completely filled with hyaline colloid masses. The contorted tubules are usually permeable, but still are sometimes similarly filled up with colloid substance. Sometimes the whole renal parenchyma is filled with bladder-like dilatations. When the greatest part of the cortical substance is thus destroyed, the vessels which supply the medullary structures and the arterioli recti become dilated and fill the latter with more blood than usual, which gives it a hyperæmic appearance.

The connective tissue in this lesion is seldom increased. There is some cellular infiltration. *Gull* and *Sutton* were the first who noticed and described arterio-sclerosis (in *Medic. and Chirurgic. Transactions*, 1872) as it exists in the shrunk kidney; afterward *Evald* and *Thoma* have thoroughly studied the morbid change in this disease, but all of these have confounded it with interstitial secondary nephritis, from which it is radically different. *Ernst Ziegler* was the first to recognize the peculiarity of this primary vascular degeneration, and has given it the name mentioned here; he has plainly set forth the great difference between this and the contracted condition of the kidney in *Bright's* disease. According to this author, the disease begins in the arterial branches, very often in the glomeruli at first, when the vascular loops become obstructed by a hyaline condensation and thickening of its walls. It develops very slowly. There is seldom albuminuria connected with it, but it may produce compensatory hypertrophy of the heart.

When a branch of the renal artery is obstructed by an embolus an embolic infarct is formed. In a very short time the whole tissue loses its vitality; it assumes a grayish or yellowish color, and great many hæmorrhages and extensive hyperæmia become disseminated in the tissue. The infarct is, according to *Litten*, produced from the capillaries by stagnation of the circulation in them and in the veins. The hæmorrhages come from the interlobular capillaries, but it is also extravasated by the glomeruli in the intracapsular space, and the tubules may also become filled with blood. According to *Conheim* and *Guilbeaux*, there may also be extravasation from the veins. The extent of the hæmorrhage may be limited or profusely spread over the whole arterial region. Under such circumstances the whole tissue will be of a uniform deep red, or spotted red and grayish white. The red color, however, soon disappears from the focal center, and the affected structures look anæmic.

The extent of the hæmorrhagic infarct of course depends on the size of the embolic artery and the quantity of tissue it supplies. It may occupy a very small spot or comprise a great portion of the cortical or medullary structures. The epithelial cells which lose their vitality shortly after cessation of the circulation become opaque, granular, their nuclei pale and indistinct, and speedily crumble. Great numbers exfoliate, become disintegrated, and form patches of detritus. The interlobular connective tissue becomes diffused, filled with blood cells and serum. The nuclei of the connective tissue become pale, the proper membrane of the uriniferous tubules becomes bloated. The glomeruli stay unchanged for quite a while, but they, too, eventually undergo morbid alteration; the vascular loops turn into solid

CHRONIC DIFFUSE NEPHRITIS.

(GRANULAR KIDNEY OF BRIGHT.)

Fig. 1.

Fig. 2.

Fig. 3

Fig. 4.

Fig. 5.

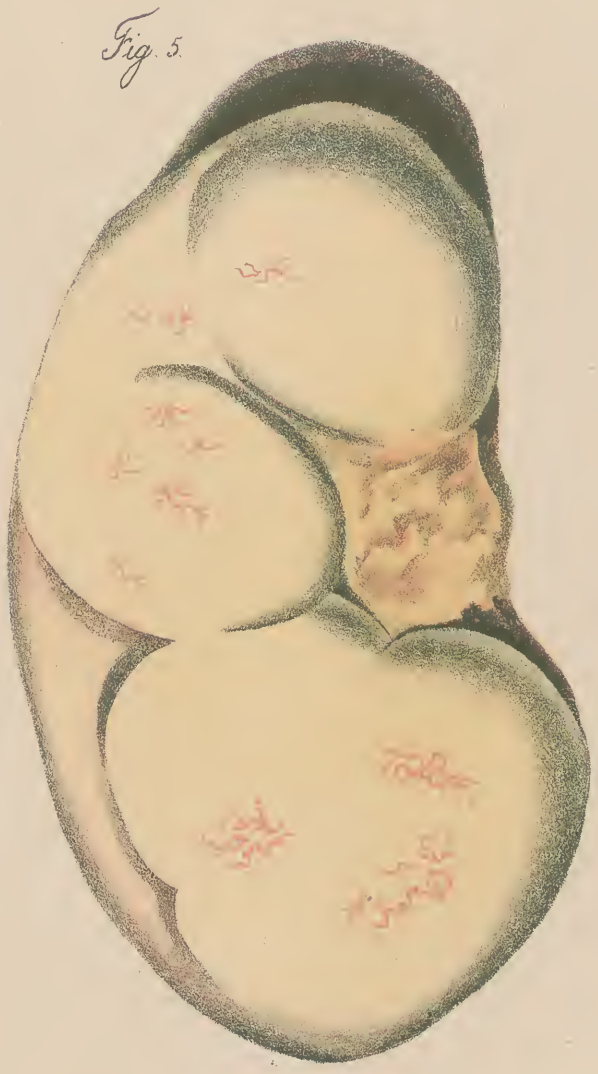


Fig. 7.

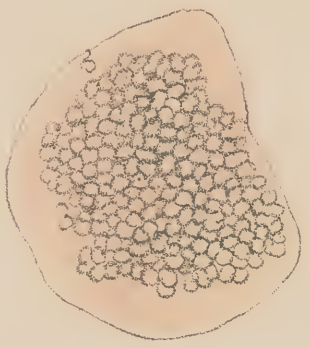
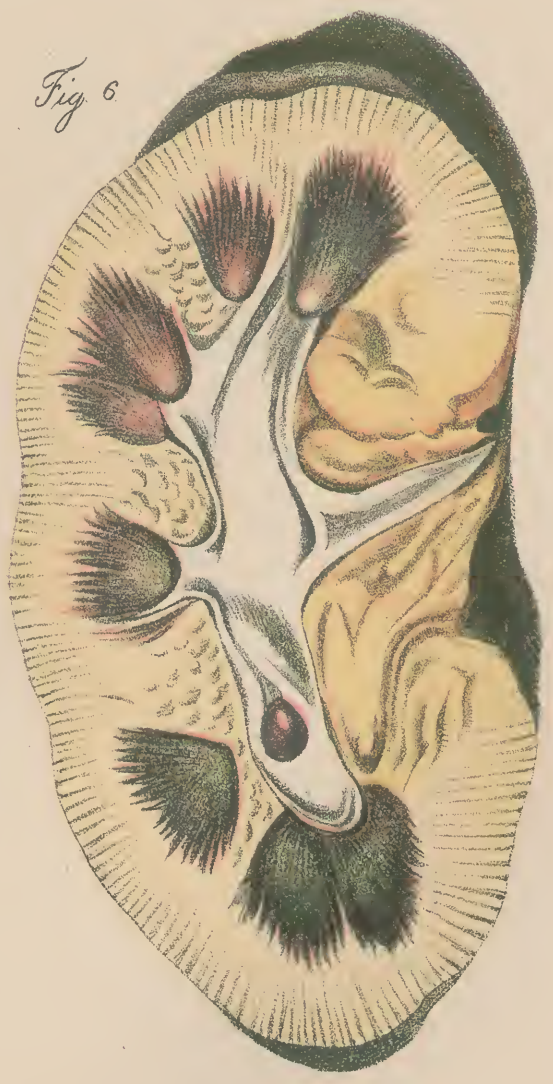


Fig. 6.



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DISEASES OF THE URINARY APPARATUS.

Sec. VIII. *Tab. III.*

ENORMOUS HYPERAEMIA IN THE KIDNEY.

ACUTE NEPHRITIS WITH FORMATION OF ABCESSES.

Fig. 2.

Fig. 1.

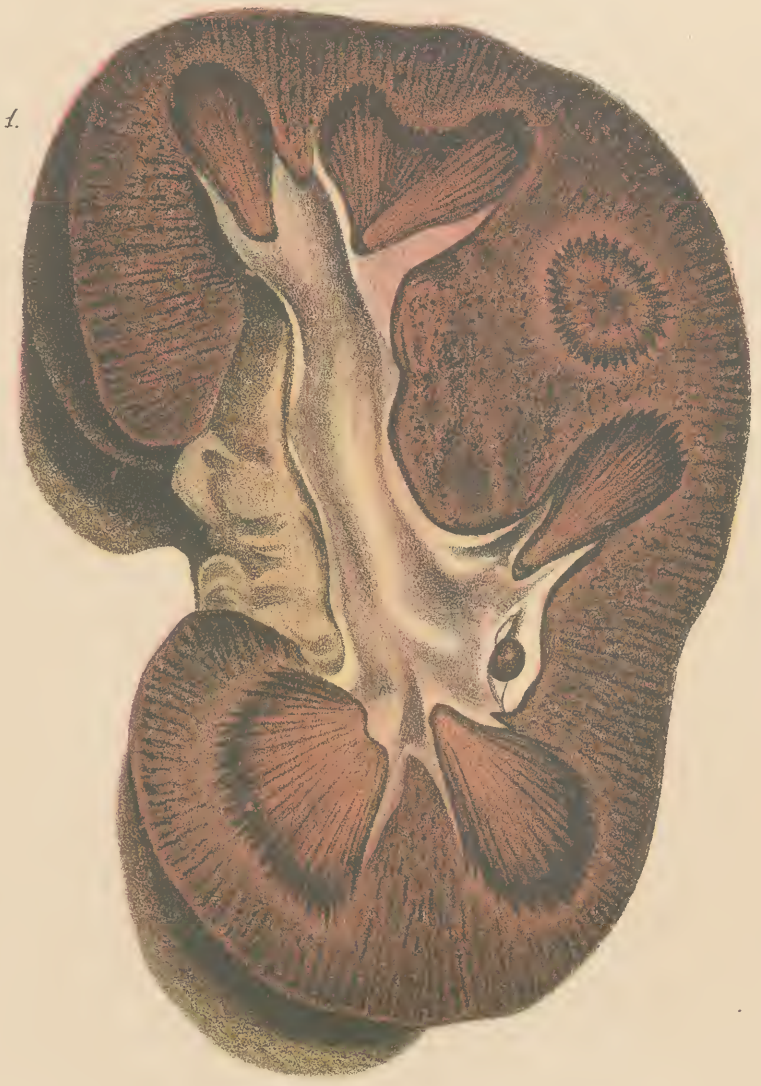

Fig. 3.

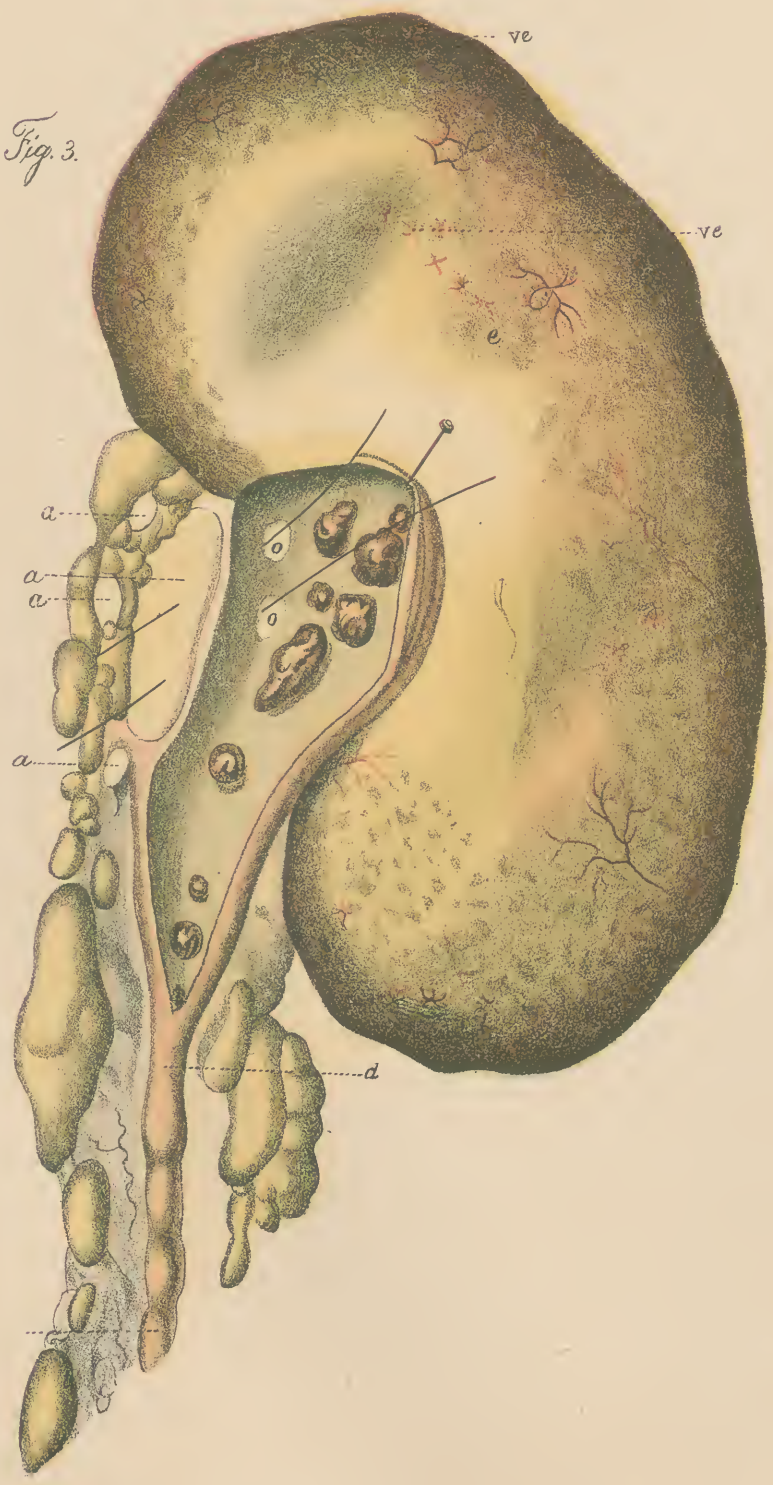

Fig. 4.

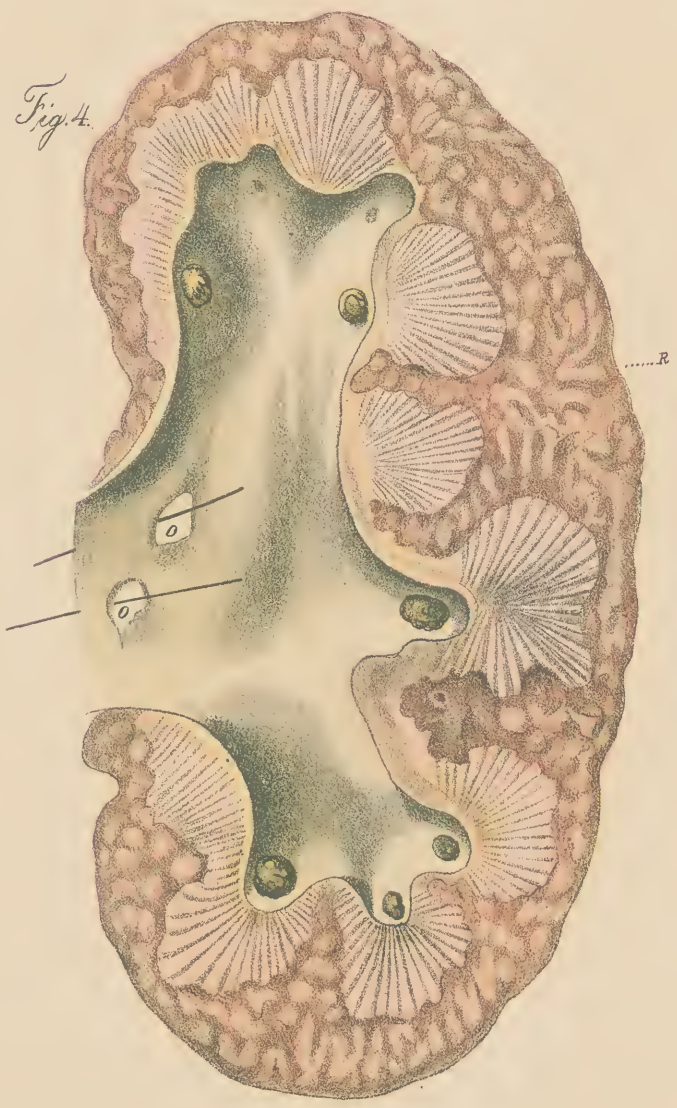

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TABLE III.

FIGS. 1, 2.—*Excessive Hyperæmia and Enlargement of the Kidney.*

CASE.—L. E., a remarkably stout man. A currier by trade his business exposed him to alternations of hot and cold. His habits were steady; was not a drinker. Ten days before admission into Guy's Hospital, London, in washing skins his feet became very wet and in the evening of that day he found his legs very much swollen, and they continued so until his entrance into the hospital. Symptoms present after admission: Urine scanty. Pulse 80. After three days treatment slight increase of flow of urine, of a dark-brown tinge, containing much albumen and a dark-red præcipitate. For several days quantity of urine increased, and of the same character. On the sixth day, pulse 84, œdema disappeared; complains of pain in the lower jaw; urine four pints, containing much albumen and some blood. Seemed to improve for several days; no blood, but heavy red sediment in rather copious urine. On ninth day complains of difficulty in swallowing; sense of constriction in throat and chest, difficulty in breathing. All treatment proved vain; he died the same night. Post mortem: No sign of dropsy; lungs gorged with blood, otherwise not abnormal. Heart and pericardium normal, liver very much congested; spleen very soft, of brown color; pulp almost liquid. Intestines normal. Bladder contains some yellow, coagulable urine. The kidneys very easily slipped out of their capsules; they are very large, rather soft, of dark chocolate color, interspersed with a few white, but a great many small black spots. A longitudinal section showed inner surface of the same color, the cortical part darker, the medullary rays a little lighter; enormous quantity of blood accumulated in the organ. The whole texture was covered with very many hæmorrhagic points. There was œdema glottidis and thickening of epiglottis. The sudden death seemed due to asphyxia from closure of the glottis. (*R. Bright's Report of Medical Cases.*)

FIG. 3.—*Acute Nephritis; Abscesses in Renal Pelvis.*

Case.—A woman of sixty years. She had suffered with asthma a great many years, and was seized shortly before death with a violent attack of dyspnœa, which being relieved she was taken with violent vomiting and deathly nausea, which speedily ended in death.

With the exception of the stomach, the inner coat of which was very dark and covered with a number of red and yellow spots, and the cavities of some intestines filled with bilious mucous, the abdominal organs, as well as the thoracic, were found normal. In the kidneys the following alterations existed: In the pelvis of the left kidney a quantity of liquid pus (A), derived from a suppurating surface situated in the outside connective tissue of the organ (O O), orifices of communication of pus with the pelvis (A A), pyogenic surfaces. The ureters and their external coverings were indurated; a number of small calculi imbedded in many parts of their walls, partly obstructing the cavities. The kidney texture very soft (R), grayish-pink outside, and covered with many stellated veins (V E).

FIG. 4.—Longitudinal section of same kidney. The internal tissue soft and friable. The whole cortical portion altered, of a whitish drab color. No infiltrations in the structures. The fine mucous membrane lining the pelvis completely disorganized. Between the pyramids deposits of thick pus. The right kidney was perfectly obliterated.

strings, the capsules collapse, and the extravasated blood is converted into brownish pigment, which stains the cylinders, often formed under such circumstances in the tubules, a deep brown. Urine secreted in such lesions always contains albumen. (*Bukmann*, *Cornil*, and *Ranvier*, *Argotinsky*, *Utthoff*, *Litten*, *Conheim*, *Weigert*, *Grawitz* and *Israel*, *Pautinsky*, etc.)

The changes which have been described only affect the central portions of the infarcted tissue. Gradually they become softened and are absorbed, particle after particle, seldom in mass. The naturally softer tissues soonest undergo disintegration, the more consistent remain longer in the organ; these generally degenerate into fatty masses or are filled with fat molecules. Others are transformed into cicatricial tissue. Very soon after the formation of the infarct most of the lesser affected parts have their circulation re-established collaterally from the adjacent cortical or even medullary vessels (*Litten* and *Pautinsky*). The balance of the injured structure recovers in a few days, either by collateral supply or by gradual opening of the obstructed vessels themselves. In extensive injuries of this kind there is never quite complete restoration of the affected part, for part of the functional tissues are either destroyed or become permanently disabled for function. Contraction and shrinking take place in the cicatrix now formed in the injured spots. They look brown, gray, or brownish red. Infarcts affecting the whole cortical portion are followed by deep furrowed cicatrices, where no vestige is left of the functional tissue, but its place is filled with connective tissue. In the parts adjacent to the totally destroyed, the functional structures are preserved in form *only*, for they impermeably fill up with newly-formed connective tissue and old structural debris, and some fat molecules. Everywhere there is diffusion of lymphoid and fusiform cellular infiltrate. The cicatricial structure is clearly defined, but adjacent to it the tubules may contain cylindrical deposits for quite a while. Only when speedy re-establishment of the circulation in the injured part takes place can recovery to normal condition take place. Usually the epithelium, even after it is regenerated, is unable to function for a long time.

According to the number and extent of emboli in the arterial region are deformities produced in the kidney from cicatricial contraction. The whole organ may thus be reduced in size, or only a part may lose its natural outline, and be distinguished by irregular depressions upon its surface.

Accumulation of Formed Substances in the Kidney and its Pelvis, derived from the Blood.

Such substances as are extravasated from the circulation either enter the renal tissue or collect in the uriniferous tubules, and pass on through the tubuli recti in the renal pelvis. From here they are carried off by the ureters or remain lodged in the pelvis for some time.

By far the greatest number of such corpuscular substances remain harmless in the renal structures. A few, however, superinduce extensive morbid phenomena and changes. Some bacteria, for instance the bacilli of anthrax, may accumulate in the renal blood circulation without causing any inflammations or other disturbances in the organ; but pyæmic micrococci bring about intense inflammatory and gangrenous processes in the kidney. *Litten* has found such quantities of bacteria in *Bowman's* capsules and in the uriniferous tubules as to choke them up completely.

Leukæmic condition of the kidneys is due to infiltration and enormous increase of colorless corpuscles in the kidney structure, in leukæmic state of the general circulation. The kidney is of a grayish white color and swells up, or it may be filled with drab-colored spots. Extravasated blood from the glomeruli, or from the interlobular vessels, produces hæmorrhagic spots and stripes of small size. The cause of such hæmorrhages may be alterations and degeneration of the glomeruli or disturbance of circulation in the renal vessels. The extravasated blood from the glomerulus undergoes decay in the capsule and leaves behind pigmentary matter in the uriniferous tubules. Both blood corpuscles and their debris, when they reach the renal pelvis, are carried away; only when great quantities of blood are extravasated in the pelvis are there formed dirty yellow or yellowish brown fibroid masses in the cavity. When blood or blood corpuscles are in great quantity destroyed in the body, a quantity of hæmoglobin or meta-hæmoglobin is eliminated in solution by the kidneys. A quantity of such coloring matter remains in the uriniferous tubules, and stained yellow and bright-colored cylinders, or casts, are found in the organ.

Precipitates of various colored biliary pigments give rise to infarcts in consequence of icterus. The kidney may therefrom assume a brown, yellow, or dark color. Only great accumulations of such substances in the smaller blood vessels give rise to great disturbances in the kidney (*Ponfick*, *Lesser*, *Marchand*, *Neisser*, *Adams*, *Bostroem*, *Luchsinger*, *Boehm*).

By transfusion of blood, especially from animals, according to *Ponfick* and *Panum*, also after extensive burns (*Lesser*) in poisoning with chlorate of potash (*Marchand*, *Lebedeff*), after sub-cutaneous injection with glycerine (*Luchsinger*), there is formed excretion of hæmoglobin not only in the glomeruli, but also in the epithelial lining of the uriniferous tubules.

By excessive production of uric acid in the body, both the acid as well as its salts may be deposited in the kidney and in the renal pelvis, in solid form. According to *Hoffmann* and *Voit*, this especially takes place when acid fermentation of the urine takes place and the presence of acid phosphate of sodium causes decomposition of urea and formation of basic phosphates. Such precipitates consist of uric acid and sodic urates; they are lodged either in the straight tubules, which they close up, or in the connective tissue of the organ. Both pulverent and granular, as well as large masses, which are described as gravel or stones, are formed in the organ. Such stones are yellow, hard, brownish, or reddish, having a smooth or a wrinkled surface. Smaller calculi usually have a crystalline, larger ones a concentric circular fibrous texture.

Calcic phosphates and carbonates form calcareous infarcts. Within the renal pelvis both gravel and calculi may be formed. Calculi of carbonate are rare; they are very hard and white. Oxalates, especially of lime, may form large crystalline calculi. This especially occurs when there is an insufficient quantity of acid phosphates of soda to dissolve the oxalates. They are in the form of yellow or brown concrements, with spinous or embossed external surfaces. Ammonia phosphate of magnesia calculi form white friable lumps or masses surrounding oxalic calculi. Cystin and abnormal constituent of urine, of yellow color, and soft, and having a radiary arrangement, are sometimes also thus formed. Formation of concrements within the kidney or its pelvis may produce inflammation of the kidney tissue (*Garrod*, *Heller*, *Neubauer*, *Salkowsky*, and *Leube*, *Charcot*, *Senator*, and *Ebstein*, *Frankel*, *Litten*).

When the epithelium of the glomeruli is very much altered, or when there is high graded disturbance of the circulation, there may be formed exudation of such components of the blood which, under ordinary circumstances, are retained in the vessels. Similarly there may be formed infiltrations from the interlobular capillaries into the tubuli. Serum is usually the liquid exuded in greater or lesser quantities; as in inflammatory nephritis when there is desquamation of the epithelium and extravasation, from the glomeruli, of serum and colorless corpuscles. The albuminates found in the urine in such diseases are not only derived from the serum but also from the corpuscular elements of the blood. Both may form part of the urinary cylinders.

Fatty molecules and other detritus are often constituents of those cylinders. Such cylinders may be voided with the urine from the kidneys, or may remain and undergo a process of softening or other changes (*Rayer*, *Arch. f. Heilkunde*, *Axel-Key*, *Schmidt's Jahrbuch*, *Vol. CXIV*, *Burkhardt die Harncylinder*, *Langhans*, *Virch. Arch. Vol. LXXVI*, *Bartels*, *Krankh. d. Harnapparats*, *Weigert*, *Weissenberger*, *Arch. f. experiment pathol.* *Rovida*, *Moleschatt's Untersuchungen*, *Sammlung*, *Huppert*, *Virch. Arch. Vol. LIX*, etc.)

Acute Nephritis or Glomerulitis. (*A.*) *Congestion, Hæmorrhage, or Diapedesis.*

The simplest form of this disease consists of an intense congestion and dilatation of the glomerular vessels. The serous exudation, diapedesis of white or colored corpuscles are phenomena immediately connected with the vascular alteration. A little later in the course of the disease the epithelium and the tectory cells swell up and become turgid.

TABLE IV.

FIG. 1.—*Phlebitis of the Renal Vein.*

This disease was observed in a woman who died during an epidemic of puerperal typhus (*Cruveilhier, Atlas Pathological Anatomy, Liv. XXXVI, Tab. V*).

The phlebitic process is exactly limited to the renal vein, passing neither beyond its entrance into the vena cava nor beyond its first subdivision in the kidney. The clot is also strictly confined to the venous trunk and not to any of its branches. In the center the clot contains solid pus. The renal structures are pale, but not much altered otherwise. (*No microscopic examination made. Ref.*)

(A A), aorta; (V R), renal vein; (V C I), inferior vena cava; (C S), clots in venous cavity.

FIGS. 2, 2′.—*Portions of the Cortical Substance Altered by Fatty Degeneracy.*

The kidney was of natural size. It seems that there has been acute parenchymatous inflammation in some portions of the cortex, and the functional structures have undergone fatty changes. The history of the case was not given by the author. His remarks on the case are irrelevant.

(B), renal pelvis; (V R), renal vein; (A R), renal artery; (U), ureter.

FIG. 3.—This is one of the rare cases of renal hypertrophy, where the whole cortical part increases in volume, whilst the pyramids either remain of normal size or develop a little more than usual. This form of hypertrophy is compensatory in its nature, that is, when a portion of the organic structure has been destroyed the remaining tissues increase in volume by enormous growth of the capillaries and dilatation of the uriniferous tubules. Quite a number of deformities are thus produced outside and inside of the kidney. (B), renal pelvis. (*Leichtenstern, Berlin, Klin. Wochenschrift*, 1881.)

Although the opportunity for examination, post mortem, of acute nephritis in the earliest stages are of the rarest, except in animals, in the process of pathological experiments, yet here and there, in very acute cases of diffused nephritis, when the organ becomes affected in different localities successively, or in very acute fevers, when the patient dies of other diseases and the renal trouble is in its earliest stage, can the various forms of hæmorrhages in the glomerular loops be studied. Such diapedic hæmorrhages may last for several days (*Cornil* and *Brault*), as experimental nephritis readily demonstrates. Great numbers of colorless corpuscles also enter into the glomeral spaces in the capsule of *Bowman*, thus obstructing the passage of urine and often producing fatal uræmia. Usually the capsule is soon cleared of these and urination proceeds more or less regularly. Whilst disturbance of the circulation goes on in the glomerulus the fixed tissues also undergo morbid changes and determine the early symptoms of acute nephritis.

B. Inflammation of the Perivascular Layer.

The simplest form of the perivascular tissue-alteration consists in a great increase of nuclei and extension of the glomeral cells into its cavity more than usual. The cells of the investing membrane of the capsule of *Bowman* swell up and assume a granular aspect. Very soon there is only found between the capsule and the glomerulus an exudate holding in suspension a few spheroid cells. Often proliferation and exfoliation of these cells take place, but which usually are soon regenerated. The inflammation of the glomerulus is always more advanced than that of the capsule. *Cornil* and *Brault* state (*Journal de l'Anatomie, Mars-Avril*, 1883, page 229) that they have never found any multiplication in the vascular endothelium in this stage of inflammation. In suppurative glomerulitis, according to the same authors, the vascular loops are invaded by a considerable quantity of lymphoid cells, derived from these loops themselves and the interlobular capillaries. These often surround the capsule of *Bowman* in several layers. In diffuse renal tubercular glomerulitis the mass of the exudated cells undergo a caseous retrogressive alteration and are suspended in a yellowish semi-colloid substance. The glomerular cavity is often dilated and the walls are compressed.

Sub-acute Glomerulitis.

1. In the ulterior stages of this inflammation in the extravascular investing membrane there is great increase of cellular elements; they either press the vascular cells into the cavity of the vessel or project from its exterior surface, or form protoplastic sheaths extending beyond the vascular wall. Accumulation of several layers of such cellular sheaths between the glomerulus and the capsule fills up the whole space between them. Gradually the vessels undergo a process of obliteration, in measure as the perivascular tissue increases in bulk.

2. Not only may cellular layers thicken the capsular wall, but also a number of actual lamellæ may form one upon the other and eventually cause adhesion of the glomerular to the capsular wall, either in single places or all over. Under certain circumstances cellular accumulations may undergo granular changes, filled with fat molecules, or with intervening fibrous bands form a sclerotic layer, which may extend to the walls of the nearest contorted tubules. In subacute glomerulitis it is characterized chiefly by violent inflammation of afferent and efferent arteries, developed in the interior of the vascular loops. Their walls become thickened and their cavities constricted. The capillaries spinning over the tortuous tubules are thus gradually obliterated, and the tubules become atrophic and are filled with fat molecules.

Chronic Glomerulitis.

In so-called parenchymatous nephritis the kidney is found, post mortem, to be very voluminous, white. The tubules, under the microscope, are seen to be dilated, filled with mucous and colloid secretions, and with all sorts of cylinders. The cortical substance is traversed in all directions by fibrous bands. Usually this inflammation is of a relatively short duration. But when the inflammatory process goes on at a slower pace and terminates in complete sclerosis of the glomerular loops, the cells in the perivascular layer gradually disappear; the few remaining are deformed, their nuclei gone, and the glomerulus is reduced to a fibrous mass. Having the ordinary contractile property of cicatricial connective tissue, it adheres sometimes to the capsular wall and sometimes it is separated from it by a clear space. From all the described facts the conclusion may be drawn that glomerulitis is as frequent a lesion as any nephritic inflammation, having a character of sufficient intensity.

Pathological Character of the Several Forms of Nephritis.

Since the time that *R. Bright* has, in very clear terms, described a number of cases of diseases of the kidneys connected with general dropsy, and which he set forth in a very detailed form in his classic *Reports of Medical Cases*, 1827 (*a number of figures are reproduced in this work*), the name of *Bright's* disease was applied at different times, by different authors, to a number of diseases of the kidneys, although to some slight extent clinically similar, yet which are essentially different from each other in the anatomical morbid alterations, their causes and especially their pathological character. *R. Bright* himself has grouped the various forms of nephritic lesions described by him, *only clinically;* all of them have one common symptom, *anuria* with *anasarca;* and one other, *albuminuria.*

Rokitansky, who wrote his hand-book of pathol. anatomy in 1842, divided *Bright's* disease in eight different forms, whilst *Frerichs* (in his work, *Bright's* diseases) classes "all the different kidney changes of persons who died of *Morbus Brighti* as only different stages of the same morbid process." According to him (*Frerich*) it begins with hyperæmia, passes through stages of exudation into and alterations of the parenchyma, and terminates in atrophy and shrinking of the whole organ. From that date a great many more or less important works have been published by authors of great reputation on the subject. Such names as *Virchow, Gull* and *Sutton, Foerster, Traube, Klebs, G. Stewart, Bartels, Kelsch, Ranvier, Charcot, Buhl, Aufrecht, Ribbert, Letzerich, Langhans, Leyden, Samuel, Eberth, Brault, Litten*, etc., should certainly imply that thorough investigation had been carried on into the subject, and that a positive opinion was formed on the nature of the peculiar lesion of *Bright's* disease of the kidneys. Yet such is not the case. Up to date there is great divergence of opinion on the subject, some of which are as follows: *Klebs* excludes from the category of *Bright's* disease all such degenerative processes in the kidney as are due to inflammatory causes, whilst he considers as belonging to that class only primary interstitial nephritis; all epithelial alterations he looks upon as secondary processes. *G. Stewart* claims three forms for *Bright's* disease, the inflammatory, the amyloid, and the atrophic. He divides the first into three stages, the inflammatory and exudative, the fatty degenerating, and the atrophic. *Virchow* classifies it into three forms, as parenchymatous nephritis, interstitial indurative, and the amyloid degenerative. *Bartels* classifies *Bright's* disease as acute parenchymatous, chronic parenchymatous, and interstitial nephritis. *Charcot*, issuing partly from a clinical and partly from a morbid anatomical standpoint, divides the disease into three forms, the first being characterized by a rapid course, scantiness of urine and highly albuminous, and constituting the large white kidney. Presence of dropsy in the whole course. The second, of slow progression, with copious urination, very little albumen, either total absence or slight dropsy, atrophy and shrinking of the kidney. The third form is the amyloid. *Weigert* considers *Bright's* diseases under two forms, the parenchymatous degenerative and nephritis proper, the first being strictly an acute disease, and the second are all of a chronic character; all are only modifications of the same process. *Aufrecht* thinks the epithelium of the uriniferous tubules becomes primarily affected, the vessels and connective tissue only secondarily. He has also three forms, the acute, sub-acute, and chronic nephritis. Nephritis includes amyloid degeneration. *Wagner* looks upon *Bright's* disease from a clinical standpoint, and defines it *as a disease in which the urine manifests certain characteristic changes;* there may be acute, chronic, atrophic, and amyloid *Bright's* disease of the kidneys. *Leyden* likewise identifies the disease with all those lesions of the kidneys which produce albuminuria and dropsy. To these also, he says, belong parenchymatous degeneration, pyelo-nephritis, amyloid changes, etc. These descriptions show the great variety of opinion of *Bright's* and other diseases of the kidneys existing among authorities.

Practically, the clinical side of the whole subject is really of primary importance, whilst the anatomical may be considered as of secondary significance. However, in morbid processes, as in all lesions of organs, the function of which depends chiefly on arterial pressure, the degree of integrity not only of the vascular wall but also of the secretory elements proper determines the exact amount of function in the organ, so will interference with this integrity also determine the degree of functional disturbance in the organ and become the indicator of the diseased state. It will manifest symptoms of the morbid conditions of the organ. The morbid anatomy will lead here to the discovery of the various clinical phases liable to exist in all nephritic derangements. *Clinically*, an acute nephritis and a chronic parenchymatous nephritis may be said to exist; also a *genuine* atrophy and shrinking of the kidney and its intermediary forms. In acute nephritis the quantity of urine is mostly diminished; it contains albumen, hyaline and granular cylinders, and as a rule also colored and colorless blood corpuscles. Still the presence of lymphoid bodies does not necessarily imply cellular infiltration, nor does absence of all cellular forms in the urine indicate that the glomerulus only, and not the tubules, are affected. Acute nephritis may terminate in death, or gradual recovery, or pass into a chronic form.

DISEASES OF THE URINARY APPARATUS.

PHLEBITIS OF RENAL VEIN.

FATTY CHANGE IN THE KIDNEYS.

Fig. 1.

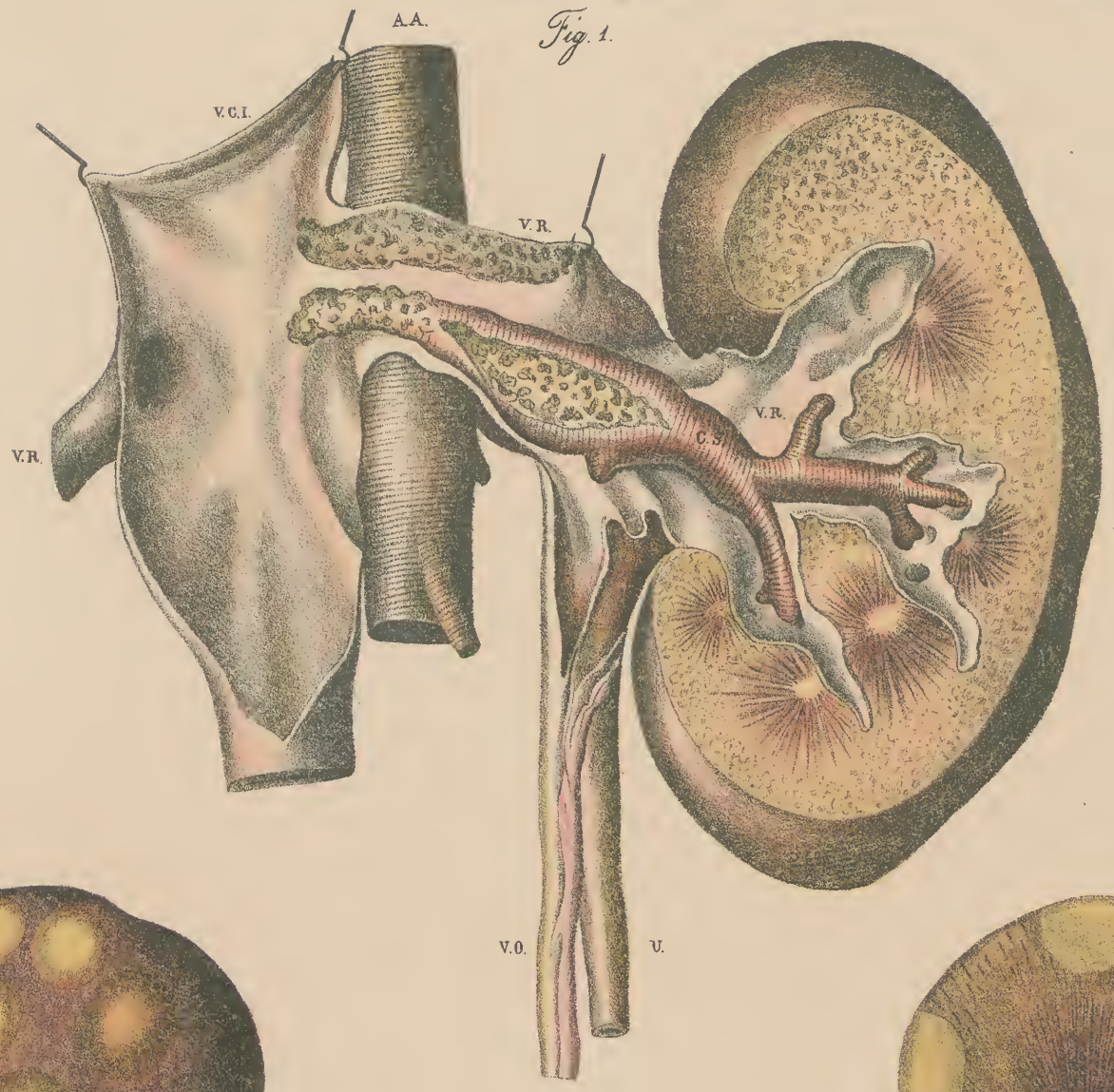

Fig. 2.

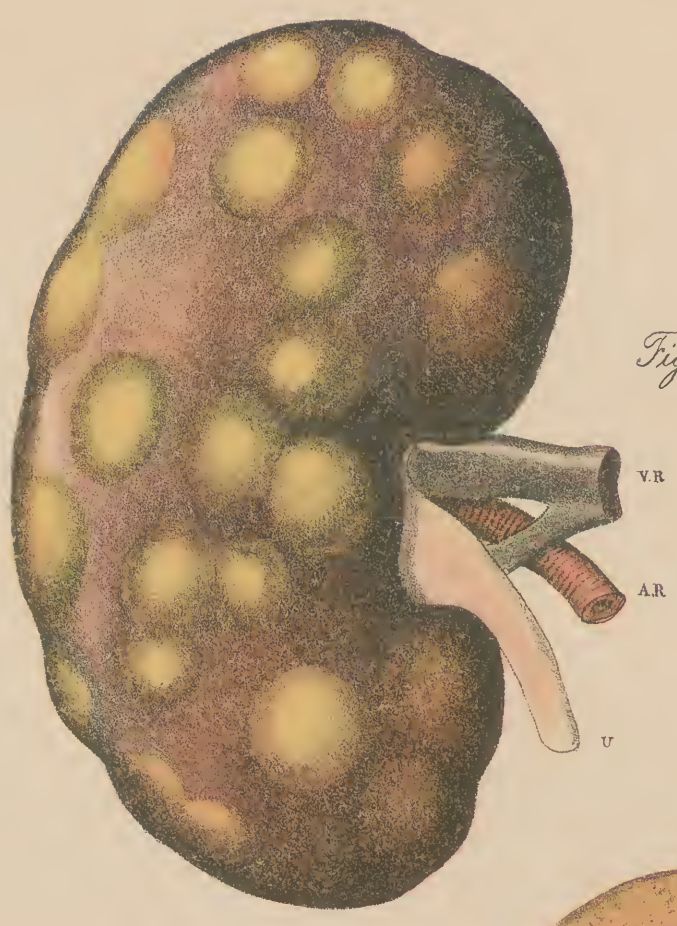

Fig. 2.

Fig. 3.

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Sec. VIII. *Tab. V.*

DIFFUSE TUBERCULOSIS OF THE KIDNEY, URETER AND BLADDER.

Fig. 1.

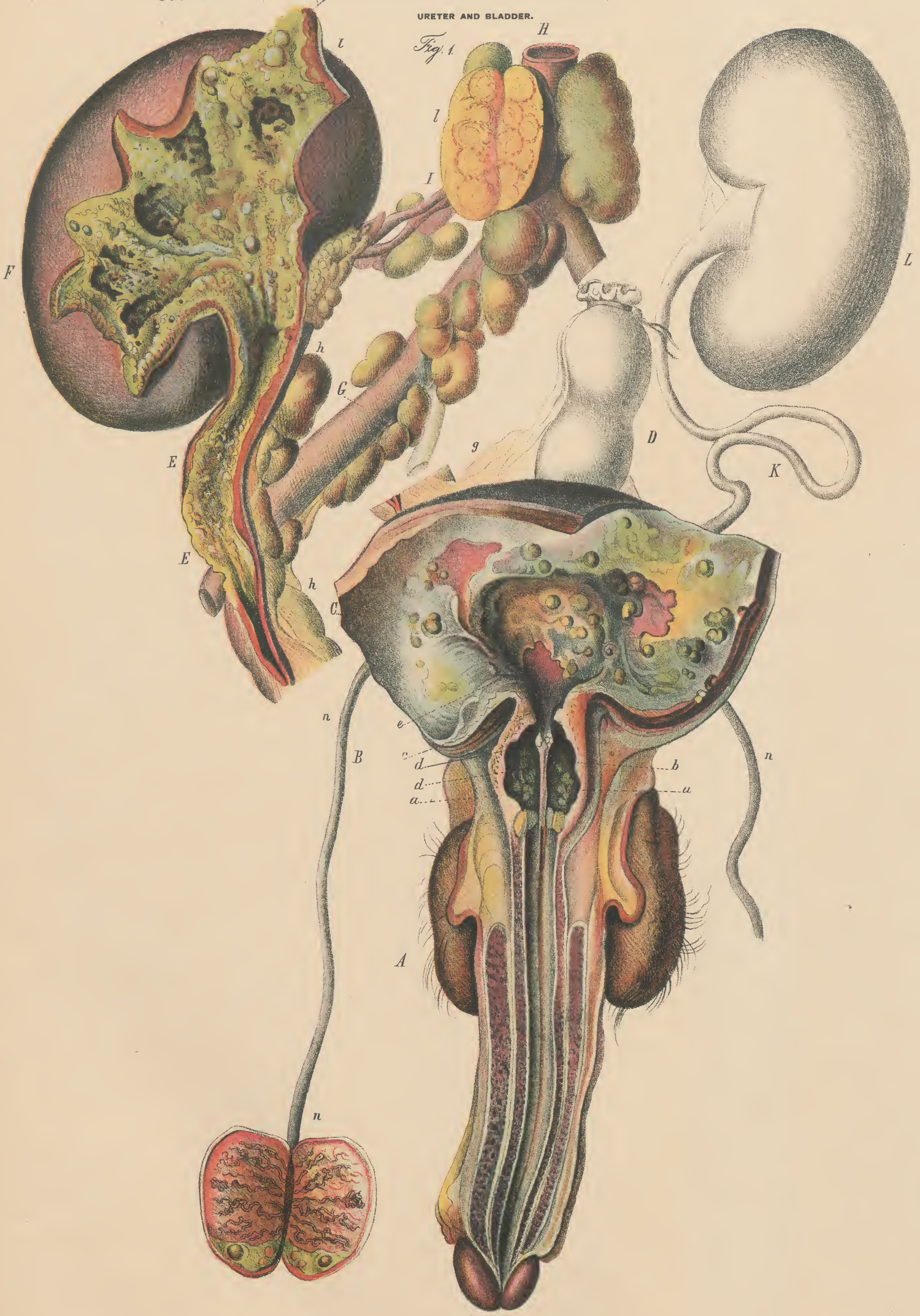

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TABLE V.

FIG. 1. *Nephritic Tuberculosis, with Ulceration and Inflammation of all Urinary Organs.*

CASE.—A man, 32 years of age.

History and Symptoms.—He is affected with hereditary tuberculous taint. Was in good health during his early youth. Became infected, at the age of 25 years, with *urethral gonorrhœa.* The disease was thought to have been cured, and he married, subsequently, a healthy young woman. Had no offspring with her. A year after marriage a mucous flow from the urethra, accompanied with great pain and constant desire to micturate, set in. Only a few drops of bloody urine would pass when he was kneeling and making great effort thereat. The passage of the little urine was joined with excruciating pains in the loins and in the back. Only great quantities of drink would produce a moderately thin film of urine. The bowels were very much constipated; the stools intensely painful. He had no rest by day or night from the distressing *strangury* and short, dry cough, with only occasional very scant muco-purulent expectoration. Two strictures, one behind the other, in the membraneous portion of the urethra, situated at the edge of the prostate gland, closed its cavity and were undilatable. The prostate gland was small and the walls of the bladder thickened. Percussion on the apex of each lung evolved a dull sound, and auscultation revealed in both apices and the middle lobe of the right lung coarse vesicular rhonchus and consonant moist rattles. After a year's treatment, in the *surgical clinic* of *Dr. O. C. Weber*, in M., during which there was some slight improvement, he finally succumbed to hypostatic pneumonia, with erysipelas. *Autopsy*, 36 hours after death. Both lungs are collapsed, and emphysematous in some portions. In both pleural cavities there is some bloody serum; also some pleural adhesions. The apex of each lung and the right middle lobe are filled with light yellowish gray tuberculous *nodes.* Some are transparent, some are granular. All are surrounded by red, inflamed tissue. The yellow tubercles are soft and consist of muco-pus; the bases of both lungs in a high state of hyperæmia; the bronchi and their branches are filled with muco-purulent thick matter; and the mucous membrane is, in many places, obliterated or softened; the bronchial glands are infiltrated with tuberculous matter and enlarged. The heart seemed rather normal; gases filled the bloodless intestines, and the mesenteric glands are all enlarged and filled with caseous masses; the liver is enlarged, spotted, and infiltrated with fat. The whole membraneous portion of the urethra is but slightly affected. At the prostatic entrance (*a*) there are a number of tuberculous lumps, which close it up (the first stricture); only a small portion of the gland (*b*) is still left; the balance forms a dilated sac, filled with highly offensive pus and tuberculous detritus (*d*). A quantity of tubercles close the entrance to the bladder (*c*); these are soft and of a bad smell; the bladder is thickened, and its cavity very much reduced; the neck and the fundus are denuded of its mucous coat, and show the subjacent muscular structure in an ulcerated and highly inflamed state, and covered with tubercles (*e*); some are soft (*f*). The left kidney and its ureter are normal; the right kidney and ureter are both enormously enlarged (F, kidney, E, ureter), and constitute fluctuating sacs; the walls of the ureter (E E) are thickened; internally it is ulcerated and covered with tubercles; the cortical portion of the right kidney much reduced in thickness, of a reddish blue color; its parenchyma dilated and covered with numerous tubercles; the calyces dilated and partly destroyed; a quantity of granular tissue surrounds the tubercles; the testes (*n*) were both normal; supra-renal capsules infiltrated.

(*A*), *Scrotum;* (*B and C*), *bladder;* (*D*), *rectum;* (*K*), *left ureter;* (*L*), *left kidney;* (*I*), *renal veins;* (*H*), *aorta;* (*G*), *common iliac artery;* (*g*), *peritoneum.*

Clinical Phenomena and Symptoms in Acute Nephritis.

A. According to E. Wagner there exist several forms of acute nephritis, each marked by peculiar clinical features and presenting special functional derangement.

1. *The Hæmorrhagic Catarrhal Form.*

The kidney is found more or less enlarged, either of normal or of slightly soft consistence, and on the external and cut surfaces numerous hæmorrhagic spots. They are disseminated both in the glomeruli and the collecting tubules. The urine contains many colored and but very few colorless corpuscles. There are sometimes very severe pyretic phenomena, sometimes but slight rise of temperature. This form usually terminates in recovery.

2. *Catarrhal Interstitial Hæmorrhagic.*

The kidney is much enlarged, generally of dense consistence. The hæmorrhages are situated in the spots as the first form and many colorless corpuscles are found between the colored. The urine contains both blood and muco-pus, and is very scanty. May terminate in recovery, or in death from uræmic complication. Now and then it may pass into a chronic form, producing compensatory hypertrophy of the heart.

3. *Medium-sized White Kidney.*

Has the color of tallow, or a little more yellow. There are no hæmorrhages. The organ is constantly in a state of anæmia. It is soft and friable. Some corpuscular infiltration exists between the glomeruli, which are compressed. Very scanty, rather clear urine. Terminates in death, either from anasarca and exhaustion or uræmia.

4. *Lymph-adœnic Form.*

The kidney is very large. All its surfaces present gray or grayish white, smaller or larger projections, white bands or rays. Very extensive infiltration between the glomeruli and the collecting tubules produces compression of these organs and suppression of urine. As a rule, this constitutes the mildest form of all, and terminates in complete recovery. It is, however, not free from danger of uræmia with speedy death.

B. According to Bartels (*Nierenkrankheiten*) true, active, renal hyperæmia exists only in the initial stages of parenchymatous inflammation of the organs, or as a result of toxic action existing in the body. Under the latter circumstances, this active hyperæmia will continue to exist in these emunctory organs until the poisons are eliminated, or until their tissues, by long continued absorption of the noxious substances, undergo a real inflammatory process. This is, however, a rare occurrence. The most frequent toxic hyperæmia is that produced by the action of cantharides or turpentine. Either of these substances may, by internal use or even absorption from the surface, call forth hyperæmia, often followed by a high degree of inflammation in the kidneys. Next to those in frequency are the noxious actions of phosphorus, arsenic, sulphuric and other mineral acids, and tartarized antimony. Though the anatomical changes in active renal hyperæmia have, from the nature of things, not yet been studied in man, yet some notion may be formed from experimental pathology upon animals. CORNIL, who has studied the action of cantharides upon animals in its different phases, describes it in the *Journal del' Anatomie* (1880, *page* 565, *etc.*) as follows: "From the beginning (*of the poisoning*) the renal tissues are deeply modified, and colorless globules are found in the glomeral capsules, although in small number. Very soon the cells of the tortuous tubules are filled with granules, and overflow with a granular liquid. Speedily all the glomeruli and tubules undergo the same process of granulation, etc." Thus the effect of the toxic hyperæmia is soon plainly manifested. Lately very numerous researches have been made into the toxic action of turpentine, and have been published in the current literature on the subject. In all cases of toxic renal hyperæmia, the symptoms appear in the latter stages as very painful strangury and constant desire to micturate. The quantity of urine is often very small; sometimes even complete anuria exists. These symptoms chiefly characterize the poisonous action of cantharides and of turpentine (*Johnson*). In poisoning with cantharides the urine collects in the bladder as a coagulated jelly, and is there retained. Pain is seldom experienced in the kidneys; the bladder and the urethra are, on the contrary, very painful. As a rule, as soon as the poison is completely eliminated, albuminuria ceases. BASHAM (*Renal Diseases, London*, 1870), however, contends that it often persists a long time afterward. Whilst albumen is always present in the urine in cases of active renal hyperæmia, blood and pus corpuscles are seldom found unless actual parenchymatous inflammation has followed the hyperæmic stage. The presence of poison eliminated with the urine is always the surest means of diagnosis in this disturbance. Renal hyperæmia alone never terminates fatally unless it passes into catarrhal or other inflammatory stages.

C. *Passive Hyperæmia of the Kidneys.*

This form of over-quantity of blood in the tissues is essentially a consecutive state of either general or special venous stasis. It constitutes a derangement symptomatic of a primary disease or lesion of the venous system. There are, according to Bartels (*Diseases of the Kidneys*), two different causes which call forth venous stasis in the kidneys, each differently modifying the renal function. (1) Stagnant conditions in the general venous system, such as are found in diseases of the lungs and heart, etc., and (2) partial venous stasis produced by obliteration or compression of the ascending vena cava above the renal veins, or of these veins themselves. The latter form may exist on one side without the patient or the attending physician ever becoming aware of it. Obliteration or obturation of both renal veins, of course, will produce very severe symptoms, and if the closure persists for some time death will speedily follow. In muscular atrophy of the heart, consecutive to pericarditis, this lesion is, according to BARTELS, most frequent, and produces well-pronounced cyanosis. Such diseases of the lungs, which produce extensive destruction of the capillaries of the lungs (for instance, excessive emphysema, or some forms of interstitial pneumonia), are the ones in which very distinct general venous stasis is to be found, and frequently passive hyperæmia in the veins of the kidneys. In all such cases this disturbance of renal function is due to reduced arterial tension, which nearly always exists under such circumstances. Such disturbances usually exist in the body a long time before any perceptible renal symptoms are manifested. Only after all possibility of formation of compensatory cardiac hypertrophy has ceased to exist, when the cyanotic color of the face and distressing dyspnœa indicate that the venous circulation in the periphery of the body is deeply hindered, then only does the patient become aware of the functional derangement of the kidneys. The urine is then of an acid reaction and very scanty; of a brown-red color; clear when just passed, but becoming speedily opaque and throwing down a heavy sediment of urates. The acid reaction is due to the presence of uric acid. Its specific gravity is generally 1,030 to 1,035. When œdema has set in the urine contains albumen, although in small quantities. Pale, slender, homogeneous cylinders are always found in small number in the urinary sediment; blood corpuscles often exist, but not in quantities sufficient to give the urine a color of blood unless some infarctus should have been formed in the parenchyma, and large quantities of blood corpuscles should pass with the water. In cases of infarct pain is felt on pressing upon the kidneys. When dropsy has once made its appearance in this class of cases, and renal symptoms with it, death usually takes place from asphyxia, but never from uræmia. In rare cases, and under exceptionally favorable circumstances, the œdema may disappear and the patient may, to a great extent, recover in a remarkably short period (*Bartels*). Such a patient may enjoy afterward relatively fair health for quite a long time.

TABLE VI.

FIGS. 1, 2. *Acute Parenchymatous Nephritis, consecutive to Acute Broncho-Pneumonia.*

The patient, a woman 73 years of age, was affected with acute broncho-pneumonia, desquamative form, ending in pulmonary phthisis. The nephritic lesion developed in the latter stage of the diseases. As no compensatory hypertrophy of the heart took place, anasarca and grave albuminuria ensued; the kidneys speedily ceased to perform their eliminatory function, and the patient died of uræmic poisoning. The post-mortem record is only of the nephritic alterations. FIG. 1—External surface, rose-colored, with very many red spots, showing diffuse hæmorrhage. FIG. 2—Longitudinal section of same, showing internal surface. The cortical substance is yellowish white, from extensive infiltration of fat molecules, within the interstitial tissue; the uriniferous tubules and glomeruli were compressed and nearly obliterated; the medullary substance was red, showing the pyramids to be in a state of hæmorrhagic inflammation; the pelvic and ureteral inner lining were pale and anæmic, but with here and there hæmorrhagic spots.

(A), renal artery; (V), renal vein; (U), ureter.

FIG. 3. *Acute Parenchymatous Nephritis, ending in formation of Abscesses.*

Both the cortical and the medullary substances were implicated (A); the pelvis and ureter laid open (B B); the cortical substance inflamed, the redness having a regular dotted appearance (C C); the same infiltrated with pus (D D); pyramids, inner portion of the organ showing small abscesses, especially near the calyces; the proper tunic of the organ is covered with dilated vessels, showing passive congestion. FIGS. 4, 5—Two cases of atrophy of the kidneys, from development of cysts in the substance of the organ. FIG. 4—(a a), right kidney; (b), renal vein and artery; (c), ureter; (d), a number of cysts, covered by the proper membrane of the kidney, and occupying the cortical substance; they were very numerous and of several sizes; (E), a single large cyst, on upper extremity of the organ; (F), a mass of fat enveloped the whole. FIG. 5—The left kidney, the organ very much reduced in size. (a a), fat surrounding the organ; (b b), renal artery and vein; (c), ureter; (E E), a multitude of small cysts in the organ, visible externally.

FIG. 6. *Suppurative Form of Nephritis.*

(a), The kidney, containing a great number of small abscesses, the result of inflammation of the cortical substance; this was consecutive to and complicated with suppurative inflammation of the prostate and stricture of the urethra; (b), enormous dilatation of the ureter, from retention of urine.

FIG. 7. *Carcinoma of the Kidney.*

(a a), Cortical substance of the organ; (B B), pyramids; (C), pelvis and ureter; (D), artery; (E), vein; (f), cylindrical mass of carcinomatous matter projecting into the renal vein, and held there by strings of connective tissue; the same occupies a number of its branches (G G G), situated within the kidney substance; (H), probe showing the partly obliterated cavities of the vessels by the carcinomatous matter.

(FIGS. 3, 4, 5, 6, 7 are from *R. Carswell's Pathological Anatomy*, as reported and drawn by himself.)

Referring each group of symptoms of each form of hyperæmia to its respective evolving cause it will be found that, although clinically similar, the morbid conditions are unlike in both, and that for practical purposes the one must be discriminated from the other. In static hyperæmia, from cardio-pulmonic trouble, the scantiness of urine is due to reduced cardiac pressure; when by any means this is raised at once *a whole group* of symptoms disappear. As the kidney tissue is here but little altered, even after long persistence of the derangement, the filtering apparatus will still carry on the work of filtration, but under very unfavorable circumstances. The products of retrograde metamorphosis may be eliminated with difficulty, but still they are eliminated, and urine laden with about five per cent. of uric acid, thick and heavy, is driven out in small quantities from the kidneys. The small quantities of albumen found in the urine are, according to *Bartels*, due to the great pressure in the renal veins permitting the serum to simply exude, mechanically, from the blood into the tubules, but are not derived from the glomerular loops. The œdema collecting in the tissues is the result of diminished elimination of water from the kidneys, and forms rather a compensatory phenomenon. Any means used to cause an increased flow of water from the kidneys suffices to stop this dropsy. In active hyperæmia the albuminates are derived from the glomerular loops, from the tortuous tubules. When great quantities collect in the urinary apparatus, as in many cases of poisoning, mechanical obstacles are placed to elimination of any urine, no retrograde products of the body pass from it; there is danger of uræmic intoxication or infarct in the kidney tissue. Further increase of arterial tension by remedial agencies often calls forth diffuse hæmorrhages. Practically, the indications for treatment would be here to remove as much as possible the obstacles in the emunctory apparatus, and chiefly the irritation from the vasculo-tubular structures. The character of the urine, as was said before, is after all the main diagnostic means by which static venous hyperæmia—cyanotic kidney—is to be differentiated from active arterial hyperæmia. In the last, the presence of blood corpuscles in the urine is almost constant, cylinders of many kinds equally so. In venous congestion presence of blood and cylinders are exceptional, and indicate complications, whilst the high specific gravity and acidity show that the disease is, strictly speaking, located elsewhere, and the disturbance of renal function is almost only symptomatic.

D. *Arterial Ischæmia and its Symptoms.*

Max Herrmann and *Overbeck* have experimentally produced arterial ischæmia of the kidneys by application of a ligature upon that artery, and have found that as long as it was contracted the urine from the kidney was scanty and albuminous. *Zielonko* found two days after the application of a ligature upon the abdominal aorta both albumen and cylindrical casts in the urine. *Loebusch* and *Rokitansky* having treated a patient with sub-cutaneous injections of muriate of pylocarpin discovered that an hour after each injection the urine contained about 75 centigrammes of albumen to the litre of liquid. They ascribed the presence of albumen to the low tension produced by that drug. They compared that condition to the one found in strong muscular fatigue, when the increased arterial activity during muscular action is followed by reduced tension of the pulse, which also becomes *dicrotic*. *Leube* found albumen in the urine of soldiers after a long and fatiguing march. *Fishl* similarly met cases with transient albuminuria in severe colic producing collapse. The urine is scant and albuminous, but contains no cylinders. This author attributes the presence of albumen to reduced arterial tension. (*Sitzungsbericht, d. K. Akad. d. Wissenschaft*, 1862. *Virchow's Arch., Vol. XLI, p.* 267. *These de Montpellier*, 1881. *François. Bericht der Naturforsch, VIII Insbruck*, 1882. *Virchow's Arch., Vol. LXXII. Stiller, Wiener, Med. Wochenblatt*, 1880. *Prager Vierteljahrs*, 1878. *Deutsch Arch., Vol. XXIV.*) Pathologically renal ischæmia is manifested in a most pronounced form in cholera, and forms what is called choleraic nephritis. *Griessinger* first called the attention of the profession to the fact that in cases of cholera there is a very scanty flow of urine, often total suppression, and this even before any perceptible change exists in the tissues of the kidneys. It is due to greatly reduced arterial pressure, or to obliteration of the renal artery. *Max Herrmann, über den Einfl. des Blutdruckes, Zeitschrift, f. rationel, Medez in 3d Series, Vol. XVII, etc. Conheim*, in his *Untersuchungen*, describes a condition of the kidneys in animals after applying a ligature to both renal arteries and veins and maintaining it for some time. He found that the functional disturbance extended to other organs besides the kidneys, and the alterations were in exact ratio to the time and extent of cessation of circulation and nutrition in the organ. When circulation was interrupted only a short time the function was speedily resumed; but when the vessels become very much dilated by long continued interruption of circulation, the tissues swell very much and become filled with cellular exudate. Recuperation then takes place very slowly. The latter fact was observed by a number of authors, the most modern of whom are *Virchow, Buhl, Ludwig, Meyer Reinhardt, Leubuscher*, and *Kelsh*. They nearly all agree as to the nature of the lesion and its cause, but differ in opinion in regard to the slowness of recovery of the renal function even after the cholera process has ceased for some time. ("The functional disturbances of the kidneys are intimately connected with the intensity and duration of the cholera process. When the disease is light it has the same effect upon those organs as any severe diarrhœa. The secretion of urine becomes tardy, and the liquid is concentrated. As long as any pulse can be felt, and the bodily heat is still retained, there is some flow of urine."—*Bartels.*) The presence of great quantities of urea in the blood of cholera patients has been observed by many older writers, such as *Hamernjek* and *Schotten*. Lately Buhl has found about eight per cent. of urea in the blood of a young woman struck down with cholera and in a state of opisthotonos. This fact explains the frequent attacks of uræmia in persons just recovering from cholera, and the sudden deaths resulting from absorption of great quantities of matter of retrograde formation, which accumulates during the cholera attack in the body from lack of renal function.

E. *Clinical Features, Course and Causes of Acute Parenchymatous Nephritis.*

The inciting causes of many cases of inflammation of the kidneys very often modify not only the intensity and duration of this disease, but also its clinical forms. Some cases begin with a very high fever, often with very violent pain others, without the presence of either. In the latter cases, the scanty urination, or sometimes its total suppression, constitutes the chief and almost sole feature of the malady. The urine is always modified in quantity containing albumen and often blood. In cases beginning with fever, vomiting or wretching and nausea, loss of appetite and other distressing febrile symptoms nearly always exist. Feverless cases are characterized by extreme debility and deep anæmia. In mild forms of the disease, such as are found after diphtheria, scarlet fever, etc., the symptoms, beyond the change in quantity and quality of urine, are often so light and so transient that the patient is usually led to believe that the inflammatory process in the kidneys has ceased; yet often proves quite delusive; for slight imprudence or neglect on his part always leads to very dangerous complications with a return of the affection. When the inciting cause is so powerful as to produce deep alterations in the organic structures, the inflammatory process passes all stages in its development, and is not only of long duration but also terminates here and there fatally. When in any of these classes of cases severe hæmaturia or anuria exists any length of time prognosis of the disease may be considered very unfavorable. Extensive dropsy, followed sooner or later by uræmia, with convulsions or coma, puts an end to the patient's life.

When the nephritic inflammation follows a less rapid course and has a less violent character, the symptoms are less pronounced. Intermittently will there now be aggravation, then amelioration of the patient's condition; but imperceptibly will the œdema, which may at first be perceived only in the face and around the ankles, spread over the body, and show his hopelessly dangerous state. Seldom is there total absence of subjective symptoms, even in the most sluggish form of this disease. In absence of uræmia, dropsy may cause the death of the affected person, either by its enormous volume

DISEASES OF THE URINARY ORGANS.

ACUTE PARENCHYMATOUS NEPHRITIS.

ABCESSES AND CANCER.

Sec. VIII. *Tab. VI.*

Fig. 1.

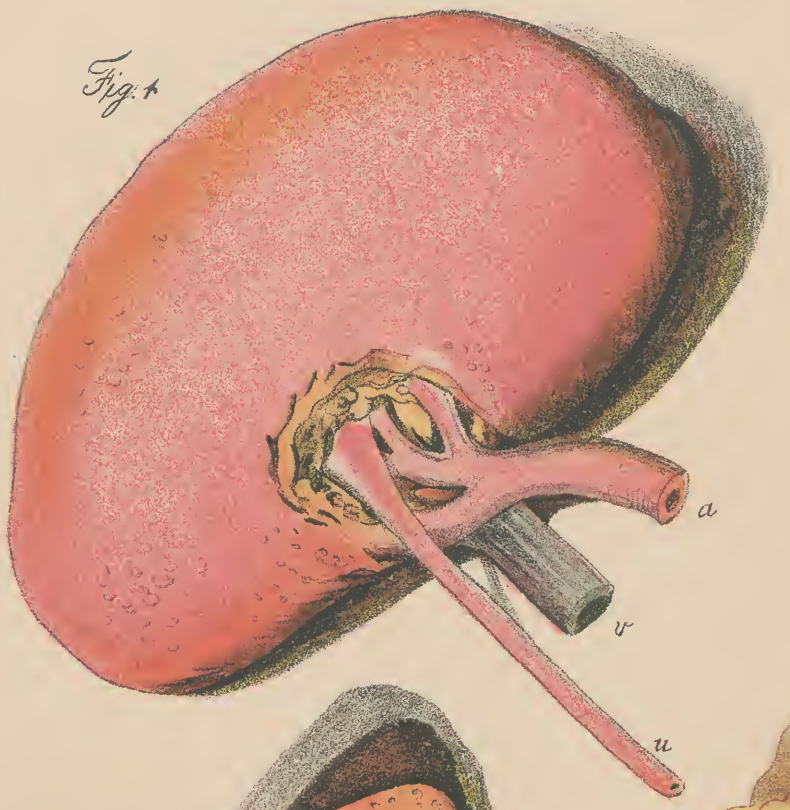


Fig. 2.

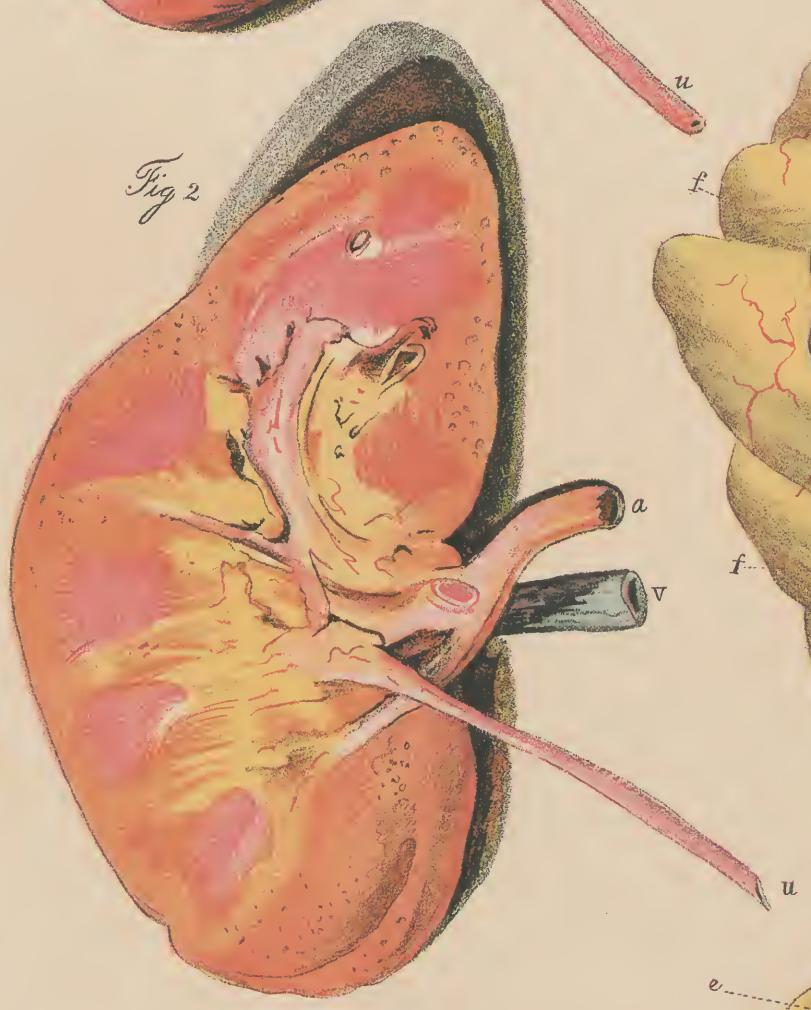


Fig. 3.

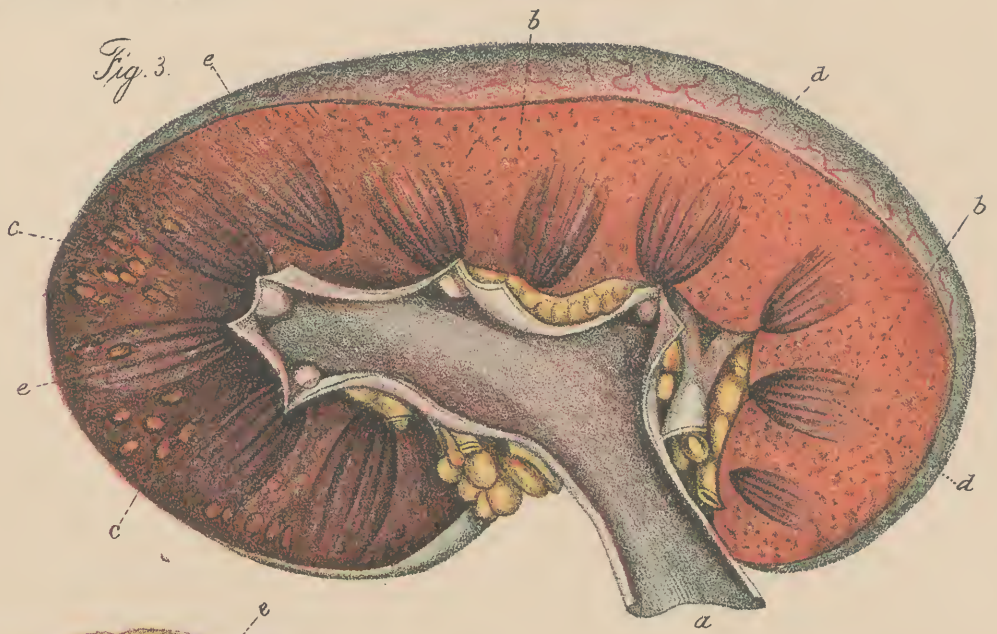


Fig. 4.

Fig. 5.

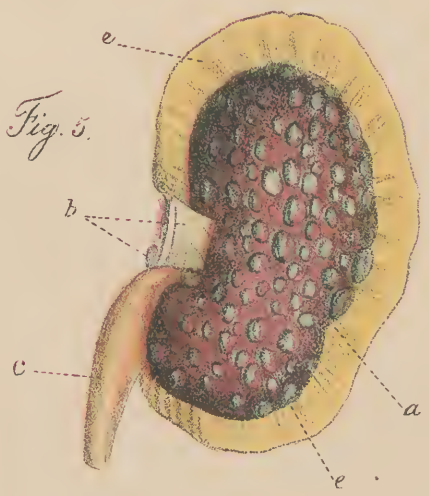
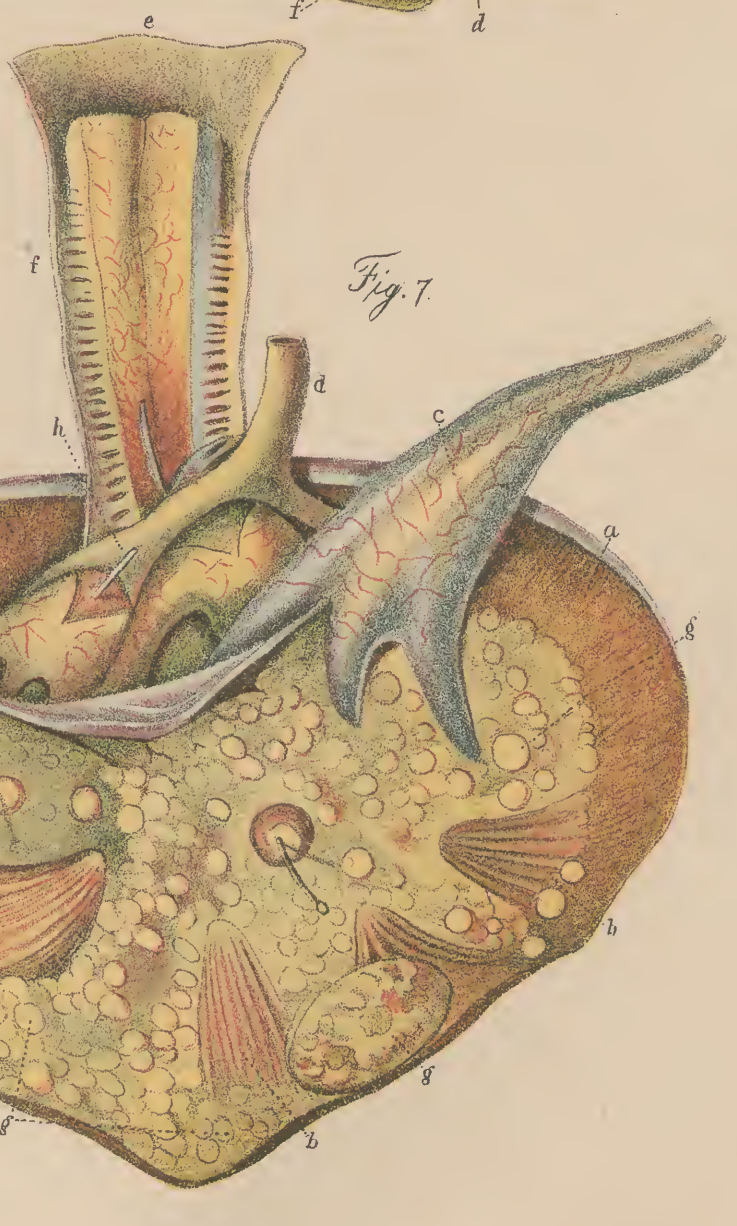


Fig. 6.

Fig. 7.



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Sec. VIII. *Tab. VII.* **PYELONEPHRITIS. PERINEPHRITIS.**

Fig. 1.

Fig. 4.

Fig. 5

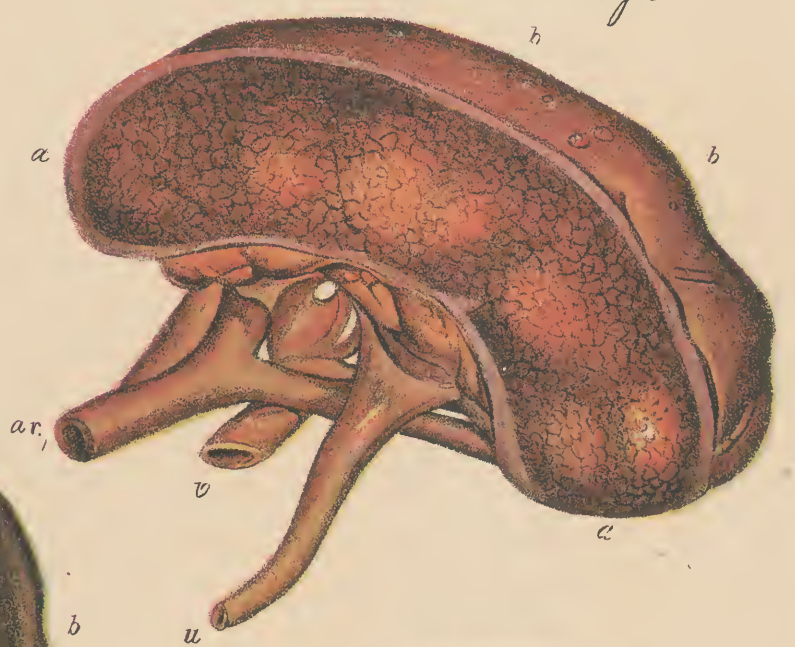

Fig. 2

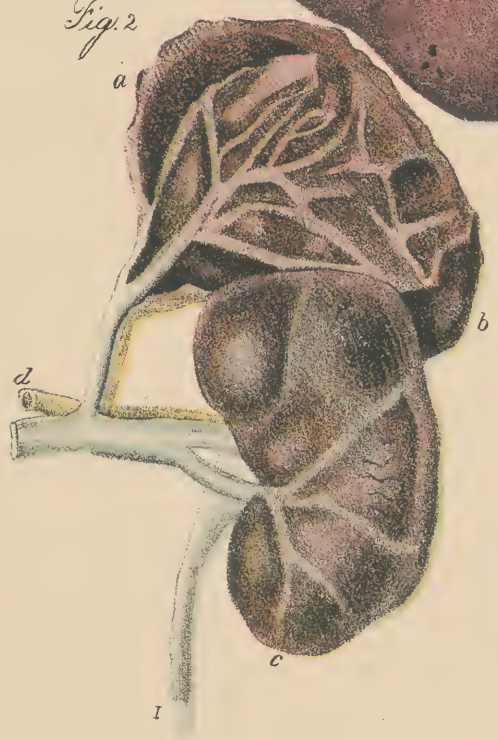

Fig. 3.

Fig. 7

Fig. 6.

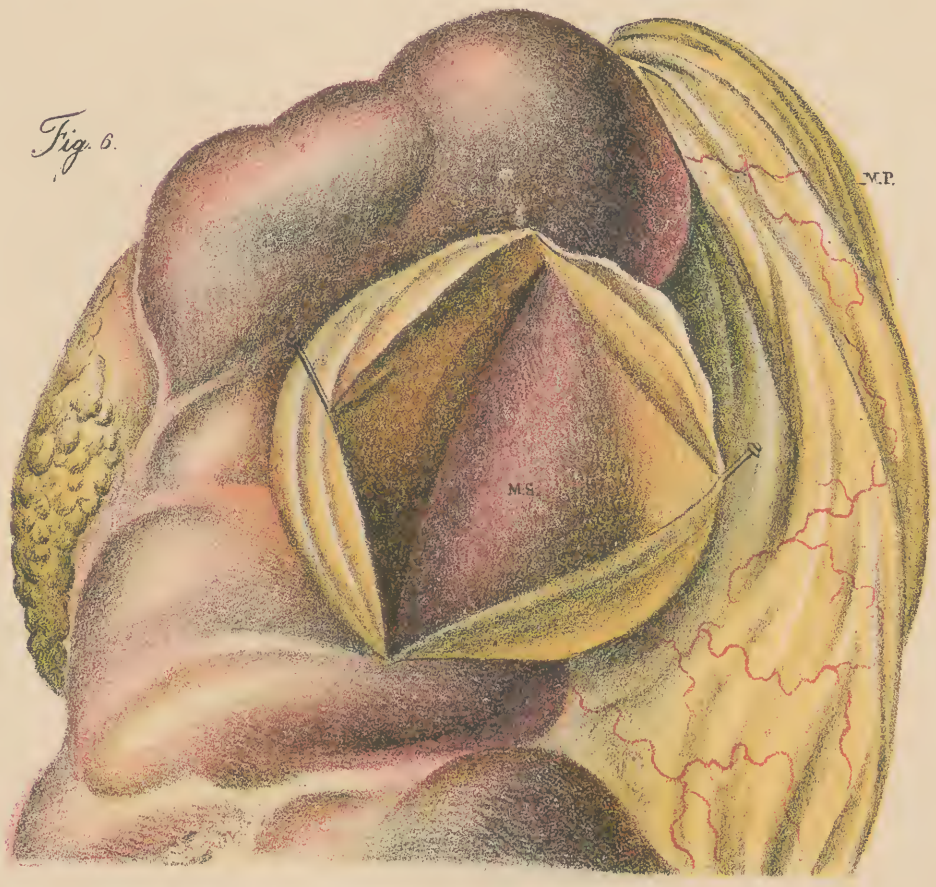

Fig. 8.

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TABLE VII.

FIG. I. *Pyelonephritis and Extensive Suppuration.*

CASE.—A woman of 30 years.

History and Symptoms.—She was confined about two years before entering the Hospital St. Pierre, and complained since that time of pain in the lumbar regions. She died in that hospital, where the autopsy took place. Post-mortem appearance: The abdomen swollen and the peritoneum inflamed, especially round the kidneys. Serous liquid in that cavity. The urine found in the bladder mixed with pus. The mucous membrane of that organ was thickened and in many places ulcerated. The urethral orifice is free. The right kidney is slightly enlarged, much pus in its pelvis, a portion of it protruding from its concave border in the form of a sack. The kidney is surrounded by a very thick pseudo-membrane outside its proper tunic. Upon the surface of the kidney, after the tunic was removed, a number of black spots, infarcts, were found. The tissue was bluish red. The lower part of organ was but a sack filled with pus, and many pockets filled with pus projected from its surface, giving it a lobulated appearance. A portion of the peritoneum and of the proper tunic were in a gangrenous state, looked green, brown, and black in spots. (A) The upper half (b) of the kidney is atrophied; (C) lower half of the organ; (D) compressed ureter; (E) renal artery small. FIG. 2—A kidney atrophied from compression produced by a very dense pseudo-membrane. It is removed from the organ, and shows fibrous bands; which enclosed the constricted kidney in several sacks. (*A C*) pseudo-membrane; (C) compressed organ. The outer surface is lobulated; (I) ureter. FIG. 3—Kidney compressed by perinephritic structures; (A b) thickened peritoneal covering, which is gangrenous in spots and perforated in numerous places. The tissue surrounding the cavities in a high state of hyperæmia (C D); brown, structureless, compressed kidney.

FIGS. 4, 5—*Fatty Degenerated and Atrophic Kidney.*

CASE.—A man of 35 years. Inebriate.

Died in St. Pierre Hospital; was under treatment of Dr. Graux. Autopsy: Anasarca and hydrothorax. The lungs but slightly changed. All cavities of the heart dilated, its walls not much hypertrophic. Muscular structure of wall yellow and filled with fat molecules. The liver, large and fatty, shows granular structure. Spleen very large and filled with blood, and dense. In the bladder blood-colored urine, forming a sediment after standing; contains much albumen and epithelium. Both kidneys in a state of granular change. Lobulated externally after removal of proper tunic. Is very soft, and seems if a number of granules are held together by a very fine network of thin fibers. In the right kidney no trace is left of any structure. In the left a slightly lighter color of the changed tissue in the places of the formerly-existing pyramids. FIG. 4—External surface of both kidneys. FIG. 5—(b b) External surface; (a a) longitudinal section showing light spots; (A r) renal artery; (v) renal vein; (u) ureter.

FIGS. 6, 7, 8—*Suppurative Nephritis.*

CASE.—Man, 63 years old, in St. Antoine Hospital. Report of Dr. Br. de Boismont. During life had much pain in the highly distended ascetic abdomen. Through the fluctuating walls a large tumor could be felt. It is very sensitive to pressure. The patient died after two days stay in the institution, from exhaustive hæmaturia, which he also had several years ago. Autopsy: Much serum in abdominal cavity. The right kidney normal, the left (fig. 6) is enormously dilated, reaching from the diaphragm, to which it is attached by its upper extremity, down into the pelvis. It is 10 inches long and about that in circumference; weighs 3 lbs. A large cyst lodged on the surface, and raising the proper membrane of the organ. (*M. P.*) The membrane is very thick, covered by tuberculous projections and many large vessels. Between it and the organ are abundant infiltrates, both of pus and serum; both liquids are contained in the cavity and dilate it very much. FIG. 7 represents a longitudinal section of the kidney. (B) Pelvis of kidney (calyees). These are very much dilated and the membrane is soft and bluish. The kidney tissue shows a network of fibers, which represent in Fig. 8 the transformed cortical substance. Between the fibers (T. R.) bloody serum and pus is much infiltrated, and a slight pressure causes it to ooze from the spongy structure. (G. R., granular structure.) This is made up of thickened and partly obliterated tubules and malphigian capsules. Of the pyramids there was no trace left.

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collected in the serous cavities, thus interfering with circulation and nutrition, by extensive œdema in the thoracic cavities and œdema of lungs, or in rare cases causing œdema of the glottis and suffocation. Pleuritis, meningitis, pericarditis, as well as suppurative inflammation of the serous tissues occur now and then as sequels of very voluminous nephritic dropsy.

By far the greatest number of cases of acute nephritis terminate in recovery. Light cases get well rapidly; grave cases, even with extensive dropsy, do often recover, only slower, of course. In the rarest of cases does the acute form pass into the chronic, and then only in such as from the beginning the inflammatory process has caused deep alteration of the parenchymatous structure.

F. *Special Symptoms of Acute Nephritis.*

Fever, when it exists in the first stages of the inflammation, may be looked upon rather as a symptom of the disease produeing the nephritis than of the kidney trouble. In some cases it is of a very high degree, and the temperature ranges above 100° Fahr. Now and then it begins with a distinct chill. A rise of temperature, in the latter part of its course, indicates deep nervous derangement and blood poisoning, such as uræmia or spread of the inflammation into other organs, and must at once engage the attention of the attending physieian. Local symptoms, such as pain over the inflamed kidney, and other subjective signs, are usually not very prominent. The pain may, in some cases, however, extend to the groin, the lumbar region, and upon the anterior surface of the thighs. The most important subjective symptoms are the desire to micturate, and the inability of passing more than a few drops at a time. It constitutes at the same time a valuable objective means of diagnosis; for as long as the nephritic process exists, there is disturbed renal function, modifying the quantity and the quality of the urine. A steady increase in quantity and natural appearance always indicate improvement in the disease. The urine looks at first more or less bloody, and when much blood is contained therein a thick chocolate-colored sediment gradually descends to the bottom of the urinal. The specific gravity in the early stages is sometimes 1,030; gradually it drops to 1,011.9. The quantity of solid elements in the urine varies with its specifie gravity. Only in the early stages are large quantities of urea contained therein; in the later stages its quantity is very small. It often happens, even when the patient is recovering, that the quantity of urine is very small. Of other elements, *Bartels* states that in acute nephritis, with general dropsy, the urine contained but few ehlorides. Albumen is nearly always present in the urine, although not in as notable quantities as in chronic nephritis. *Henoch* and *Ferini* state they have met with cases of nephritis without albuminuria. Blood, likewise, is most frequently found in acute nephritic urine; sometimes it constitutes its earliest symptom. The quantity varies greatly, and is not always in proportion to the quantity of albumen. Albuminous urine, of acute nephritis, always contains cylinders, yet varying greatly in quantity; the kind of cylinders depends on the stage of the disease. Besides these, colorless and spheroid corpuscles in different stages of alteration are generally found with desquamated epithelium of the urinary tubules, in various stages of disintegration. The cause of diminished secretion of urine in inflamed kidney is as yet not quite clear; it is undoubtedly due to some vaseular alteration, but so far the exact cause is not known. The danger attending acute nephritis is owing to grave functional disturbance in the organ, especially in its earliest stages. The products of tissue-decomposition are retained in the blood and in the tissues, when the flow of urine is checked and ultimately arrested. These give rise to poisoning of the body with its waste product; besides, in this state very dangerous suppurative inflammation readily develops in the several serous cavities of the body. Lack of elimination of water from the body gives rise to œdematous infiltration in the tissues, with all its consequences.

G. Desquamative nephritis, or catarrhal nephritis, is seldom a primary disease. Taking cold and getting wetted through, is generally considered the cause of the primary form. Whilst it is undoubtedly true, as very numerous well-authenticated cases of the kind by very reliable authors have been reported, that sudden cooling of the bodily surface is certainly liable to produce very severe derangements of the bodily functions, the exact *modus operandi* upon the organs, and in which way the eooling produces permanent diseases, or even lesions, is as little understood in primary nephritis as when the same accident is said to have produced acute pneumonitis. The commonest cases are secondary in their nature; they may be due to ascending inflammatory processes from the inflamed mucous lining of the ureters, bladder, urethra, etc. The catarrh progresses from the renal pelvis to the papillæ, and when the urethra-vesicular phlegmasia is light, the kidney trouble will persist longer than the original disease. (*Jaccoud, traité de patholog. interne.*) Poison of different kinds, as was stated before, produces several forms of this catarrhal trouble. *Virchow*, *Reinhardt*, and *Schroff* have established the fact that in this disease there is a distinct fluxion and exfoliation of the epithelial structure of the kidneys. The researches of *Thoma*, *Runeberg*, and *Litten* have shown that in fevers, when there is diminished pressure in the blood vessels, with dilatation of the vascular loops of the glomeruli and stagnation of the circulation therein, or even reduced pressure of the tubules, albuminuria soon ensues. The prineipal febrile diseases usually found complicated with nephritis are typhus, enteric typhoid, parenchymatous atrophy of the liver, sometimes small pox, measles, and erysipelas. In scarlet fever the kidney trouble usually makes its appearance during or shortly after the cutaneous exfoliation, in typhus after the third week of the fever, and in typhoid it may be observed from the beginning, in diffuse hepatitis during the second or the dyscratic stage (*Jaccoud*).

A. *Chronic Parenchymatous Nephritis, Bright's Disease, Interstitial Nephritis, etc.*

That morbid complex, discovered and described by *Bright* (in his *Reports*), consists of the following phenomena: *Persistent albuminuria, dropsy having a special course, and a lesion of the kidneys.* In all cases of the kind the clinical features are all alike, not so the morbidity of the anatomieal elements. It is now almost universally conceded that there are three forms of the disease: (1) parenchymatous nephritis, the commonest of all, and known as the large white kidney; (2) interstitial or sclerotie nephritis, the small red kidney, also named granular atrophy; (3) the mixed form, partaking of both. Simple catarrhal nephritis, which gives rise to only temporary albuminuria, has nothing in common with Bright's disease.

The commonest form, the *epithelial* or diffuse parenchymatous nephritis, is generally eaused by *severe sudden* cold-taking, or by slow and gradual impression made upon the body by damp, dark dwellings; infectious diseases, such as scarlet fever, typhus, very severe attaeks of small pox, especially of the hæmorrhagic kind, measles sometimes, infectious endocarditis, and puerperal diseases. The two latter are the most liable to give rise to nephritic lesions, intermittent and paludal fevers, severe forms of malaria. (*Jaccoud, pathologie interne.*) *Bamberger* has found that of 2,430 cases of

TABLE VIII.

RENAL LITHIASIS.

FIG. 1 represents a urinary calculus contained in the renal pelvis. The kidney is atrophic, its color is yellow; the external surface is covered with dilated vessels; the atrophy is due to pressure exerted by the calculus upon the kidney. FIG. 2—A remarkable case of commencing lithiasis. There is but little change in the organ beyond its yellow coloration by urinary coloring matter; the cortical substance is somewhat granular, but the size of the organ is normal. (*C*), calculus; (*S C I*), injected coloring matter. FIG. 3—Urinary calculus in another kidney. FIGS. 4, 5, 6—*Renal Lithiasis*, the case reported by Dr. *R. Froriep*. The person affected was a man 54 years old; had suffered for years with intermittent fever; had lately complained of pain in the plantar surface of the right foot; was suddenly attacked with fever and delirium; the big toe and the dorsum of that foot had been frosted years ago, and now a black blister, containing yellow, odorless liquid, had formed there, and was surrounded by highly inflamed tissue; the whole foot became swollen and erysipelatous; the big toe swelled enormously. Notwithstanding the careful treatment by *Dr. Diffenbach*, the man continued to grow more and more feeble; he became speechless; his pulse grew exceedingly feeble and very frequent, his breathing short and superficial; the tongue was covered with a brown coating and very dry; the abdomen very tender, and watery stools were involuntarily discharged from his bowels; fibrillar tremor and unconsciousness set in, and the third day after admission into the hospital he died in a comatose state. Autopsy, shortly after death. Very extensive chronic ulceration and sloughing had destroyed much of the tissues of the right leg and ankle; in the cranial cavity everything was nearly normal, with the exception of some serous infiltration in the arachnoidal cavity; in the chest both pleuræ were found inflamed and covered with fibrinous exudations; much liquid in both cavities; the heart slightly dilated, but not altered; in the abdominal cavity, all normal but the kidneys, both of which were equally affected. FIG. 4 represents the appearance of the inner surface of the right kidney. The organ is divided, by a longitudinal section on the convex border, into two equal halves, and is held together by its concave border; the renal pelvis and the beginning of the ureters are thus exposed. There is but little change in the lining and arrangement of these cavities; the calyces are divided, and show their anatomical relation to the pyramids. There is considerable of fat between the pelvic structure, the calyces, and the renal structures. A portion of a pyramid is enlarged, and shows in Fig. 5 (a) deposits of whitish yellow calcareous matter in the intertubular spaces; (b) a good-sized phosphatic calculus is lodged in the extremity of the organ, and has, by causing ulceration, destroyed a portion of the renal substance (c).

FIG. 6—External surface of the same kidney, where the abscess has perforated the tissues and caused depression. FIGS. 7, 8—*Urinary Organs in Cholera*. FIG. 7—A section of kidney of a person shortly after death from asphyctic cholera. The renal pelvis is divided; the substance of the kidney is of a darker color than normal; the pelvis was found empty, and even the odor of urine did not exist; the pelvic lining was pale and covered with bluish veins.

FIG. 8—The bladder of the same person; it was contracted to a small size. (A), fundus; (b), external surface, separated from its surrounding connective tissue; (C), neck of the bladder wrinkled, which makes its walls look thicker than they really were; very numerous hæmorrhages, and congestion of the bladder and prostate; their mucous membrane was pale; a quantity of yellow mucus was lodged between the folds; the neck is somewhat injected, and the prostate is full of hæmorrhagic spots.

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syphilis under his observation, two per cent. were affected with Bright's disease.

A great many other pathological conditions of the body may lead to the development of Bright's disease, partly by continuance of inflammatory processes going on in adjacent organs or in those with which the kidneys are physiologically connected; partly by retention within the body of excretory substances; partly also from lesions and diseases of the organs of circulation and respiration; many dyscratic conditions of the body, tuberculosis, cancer, etc.; a number of poisons, when they are slowly eliminated from the body and keep up a state of irritation of the kidney tissues, also lead to development of some forms of Bright's disease. *Munk*, *Leyden*, and *Mankopf* have shown that sulphuric acid intoxication also causes the same form of nephritis. According to *Jaccoud*, the disease develops in different countries and climates from different causes. In England it is mostly brought about by gout; in Scotland, according to Christison's report, it forms a complication with alcoholism; in Sweden and Norway, where alcoholism is most frequent, Bright's disease is very seldom caused by it; in France, miasmatic diseases seldom produce this affection; *Frerrichs* reports that it is very rare in Breslau, whilst *Rosenstein* reports it to be very frequent on the shores of the Baltic sea. It seems that the same causes are of unequal potency in different localities (*Heidenhein*). The many and various causes which give rise to simple albuminuria, *Jaccoud* (*pathologie interne*) has classified as follows: (1) modification of the mechanical conditions of kidney circulation; (2) alteration in the composition of the blood; (3) alteration of the same with renal lesions; (4) lesions of the renal tissue only. As regards the causes of albuminuria of Bright's disease, the same author recognizes only such where there is pathological alteration, either functional or anatomical, of the secretory apparatus, that is, the *glomerular apparatus*.

B. *Interstitial Nephritis, Renal Sclerosis.*

A few of the above-named causes may also give rise to the indurative form of Bright's disease, with this difference—that this form has never an acute beginning, but has from the very first a chronic course. The best ascertained of its causes are organic lesions of the heart; many other assignable causes are still not settled among authorities. *Lancereau* has, however, thoroughly established that alcoholism may give rise to granular kidney. As a rule men are far oftener affected with Bright's disease than women. From the age of fifty to sixty-five it gains in frequency. Comparing the different forms of nephritis affecting persons of different ages, *Jaccoud* found that early childhood and youth are subject to more acute forms, to chronic, etc.; chronique parenchymatous mostly from the ages of thirty to sixty, whilst the interstitial atrophic prevails from the ages of forty to seventy years.

C. *Clinical Features of Bright's Disease.*

The parenchymatous form, when commencing as an acute inflammation, is characterized by a chill, fever, with pain in the loins, vomiting, and followed in a few days by general anasarca, which, under favorable circumstances, disappear after the third week of its course. Examination of the patient's urine is the only sure differential diagnostic means to decide between this and other high-graded fevers and inflammatory diseases. The cardinal symptoms may be classified as alterations of the urinary secretions, dropsical effusion, and alteration of the constitution of the blood. In the clearly acute stages, the urine has most of the physical properties common to other forms. It may be reduced to one-half or even one-fourth the average normal quantity (900, 700, 500 grains); be of an acid reaction and varying tendency, between 1,025 and 1,045 sp. grains. The color is of a more or less deep red, from the greater or lesser quantity of blood corpuscles (not hæmaticæ only), which fall to the bottom of the vessels after standing awhile. Albumen is always present, sometimes in astonishing quantities. The coagulum is of a brown color. Under the microscope are seen *renal epithelium*, *blood corpuscles*, *real fibrinous cylinders*, *and especially lymphoid bodies* (TRAUBE, RICHARDSON). After cessation of hæmaturia, the fibrinous cylinders gradually disappear, and cylinders composed of epithelial debris with some albumen appear. This urine contains far less area and phosphates than ordinary febrile urine. The daily loss of albumen by the kidneys varies from 5 to 25 grammes (*Frerrichs*). The typical urine of the acute stage always passes into an intermediary, which is characterized by its quantity being nearly normal, of acid reaction of 1,022–1,012 density; urea, uric acid, chlorides, and phosphates diminish in quantity; daily loss of albumen very variable; its presence is intermittent, more during the day than in the night; morphological elements are epithelial cells, fibrinous cylinders, mixed cylinders of albumen, epithelium, and colloid masses. Presence of blood corpuscles indicate past hæmorrhage from the first stage.

D. *Sub-acute Beginning of Bright's Disease.*

In many cases the initial stage is so insignificant as not to become perceptible until dropsy has set in. Sometimes very severe lumbar pains call the attention of the patient, and are often mistaken for lumbago. They increase when the affected person walks or stands for some time, by upright or sudden movements of the back, by bending forward, or by pressure on the region of the kidneys; such pains often appear before the dropsy. Occasionally persistent laryngo-bronchial catarrh and disorders of vision will engage the attention, which, when the urine is examined, is found loaded with albumen, and the opthalmascope indicates *albuminuric retinitis*. Now and then persistent diarrhœa, vomiting, very rapid loss of flesh, or epistaxis will awaken suspicion of existence of the disease. *Dieulafoy* noticed some cases of this nephritis which began in sudden deafness; likewise may persistent sleeplessness, with repeated desire to urinate and scantiness of urine, indicate the beginning of the disease. The character of the urine in such cases is like that existing in the second stage of the highly acute type.

E. In the distinctly chronic type the urine is pale; the foam produced in letting the urine will stand for quite a while; reaction slightly acid; the quantity very variable, not plentiful at first it diminishes gradually; uræmia is indicated by suppression; the density gradually lessens, and may drop to 1,004 specific gravity; there is then nearly total lack of elimination of excretory substances by the organ; the quantity of urea is very slight, albumen very variable; it is usually quite absent in renal atrophy; globulin and peptones are often contained in the urine. *Petro* found in 41 cases 28 of peptonuria and 13 globulinuria.

F. The principal morphological elements in the urine are fatty granular epithelium, and cylinders which present the chronic type in three different forms: hyaline cylinders, yellow or waxy, and granular. The hyaline may appear both in the acute as well as in the chronic stage of diffuse nephritis; they are, according to *Rovida*, of a peculiar proteid substance; they are covered with blood globules and epithelium. The yellow or wax-like variety and the granular are modifications of the same morbid structures; their presence indicate the chronic form of diffuse nephritis, and point to a grave condition of the organs.

G. The change in the constitution of the blood in the acute stage is an increase of fibrine, which is peculiar, according to *Frerrichs* and *Christison*, to all forms of phlegmasia. In the chronic form, the alteration is due to derangement of function of urination; the density of the serum drops sometimes to 1,020; the quantity of albumen in the blood diminishes in the ratio to the loss through the kidneys; the serum undergoes a physical change, and much of it circulates in the body in the form of coagula; it presents a milky and turbid aspect, which *Christison* and *Rayer* have attributed to the presence of free molecules, but which *Simon*, *Scherer*, *Buchanan*, and *Sullivan* have proved to be due to those suspended albuminous particles, which *Frerrichs* has perfectly verified. In the beginning the red and colorless preserve their

RENAL LITHIASIS.

KIDNEYS IN CHOLERA.

Sec. VIII. *Tab. VIII.*

Fig. 1.

Fig. 3

Fig. 2

Fig. 5

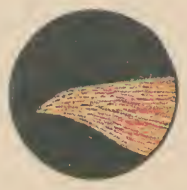

Fig. 7

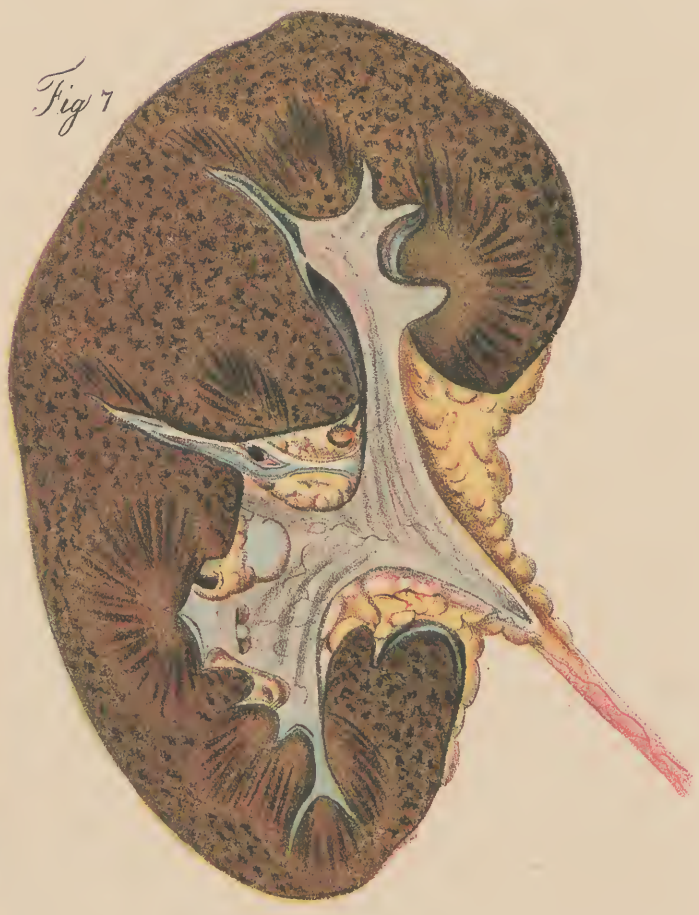

Fig. 4.

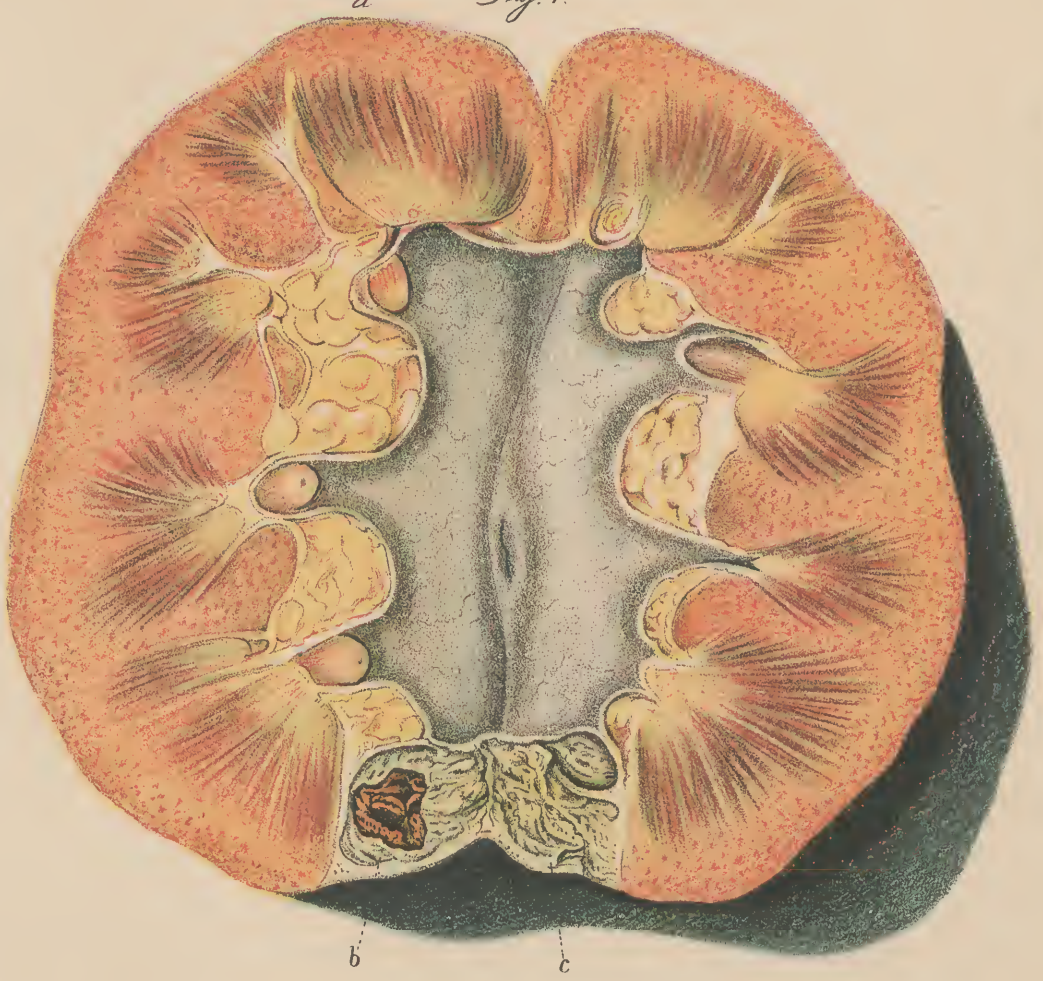

Fig. 8.

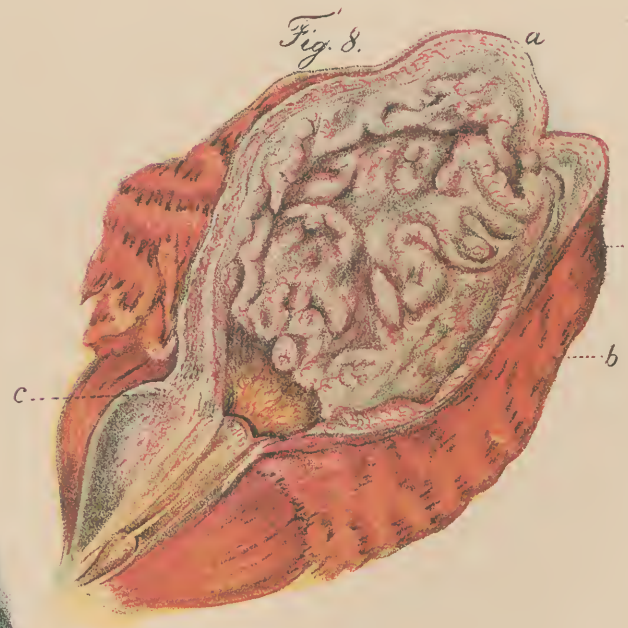

Fig. 6.

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Sec. VIII. *Tab. IX.*

CHRONIC NEPHRITIS,
INTERSTITIAL NEPHRITIS.

Fig. 1.

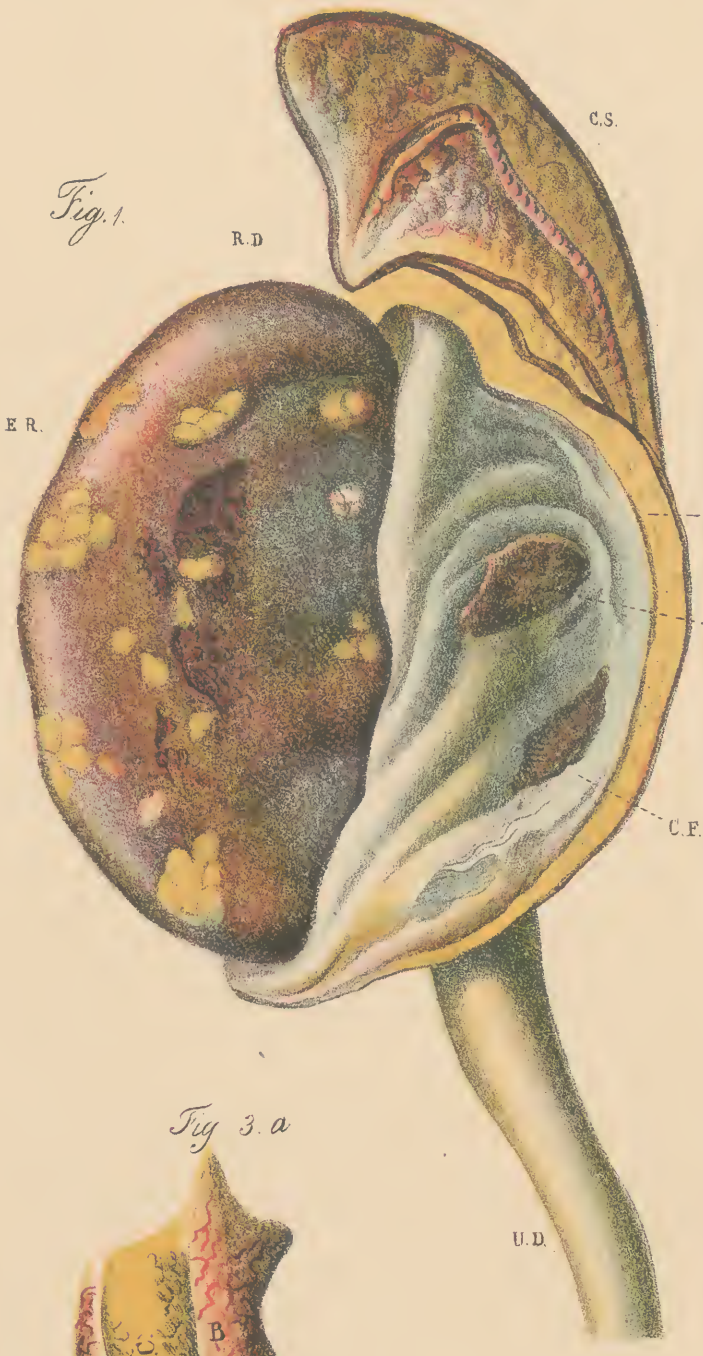

Fig. 2.

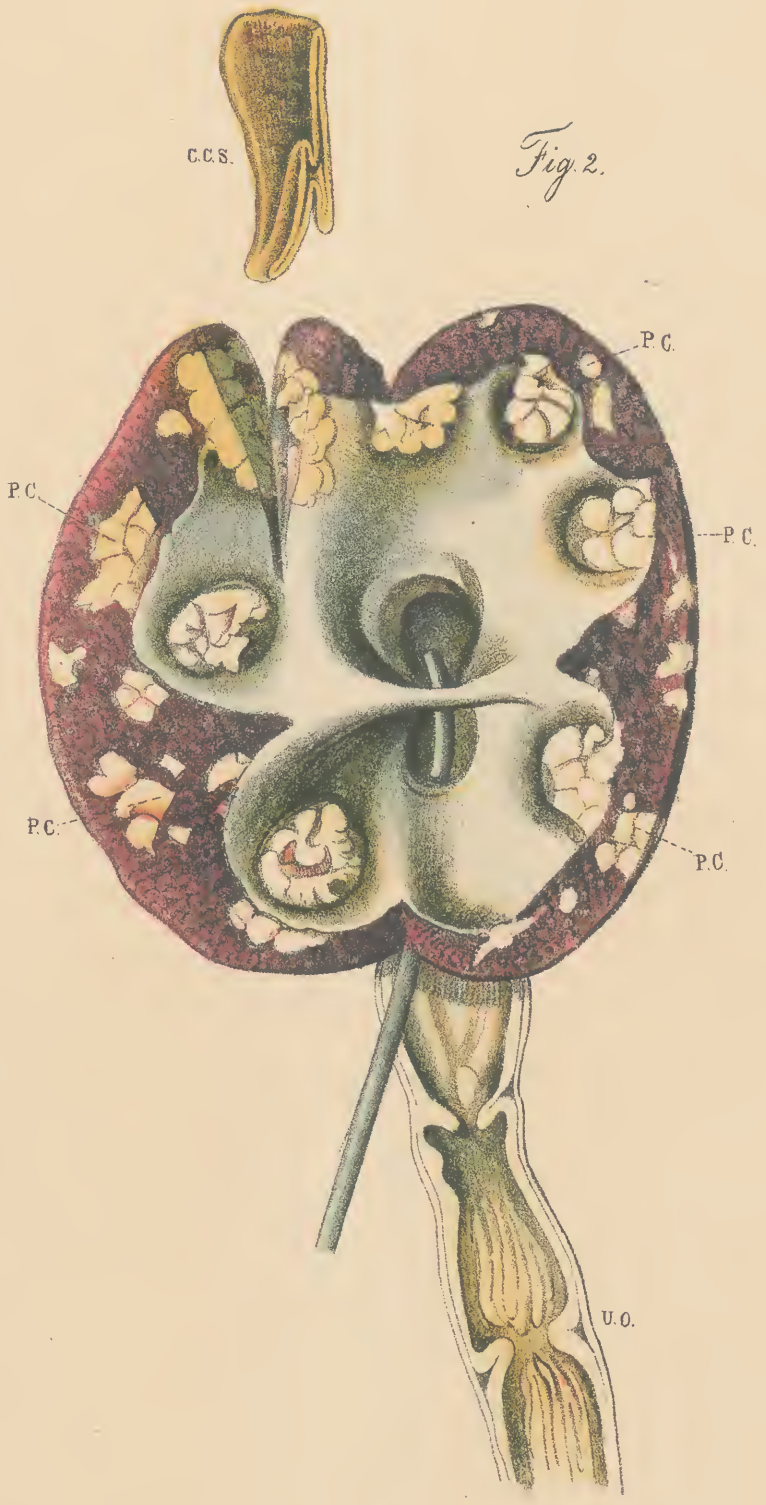

Fig. 3.

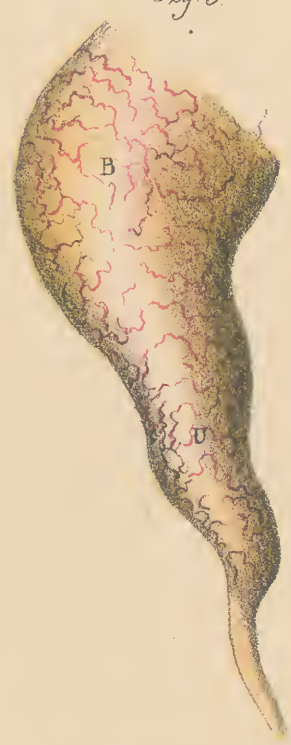

Fig. 3. a

Fig. 4.

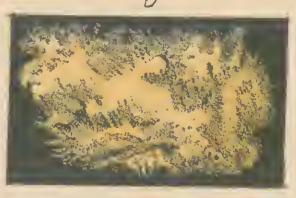

Fig. 5.

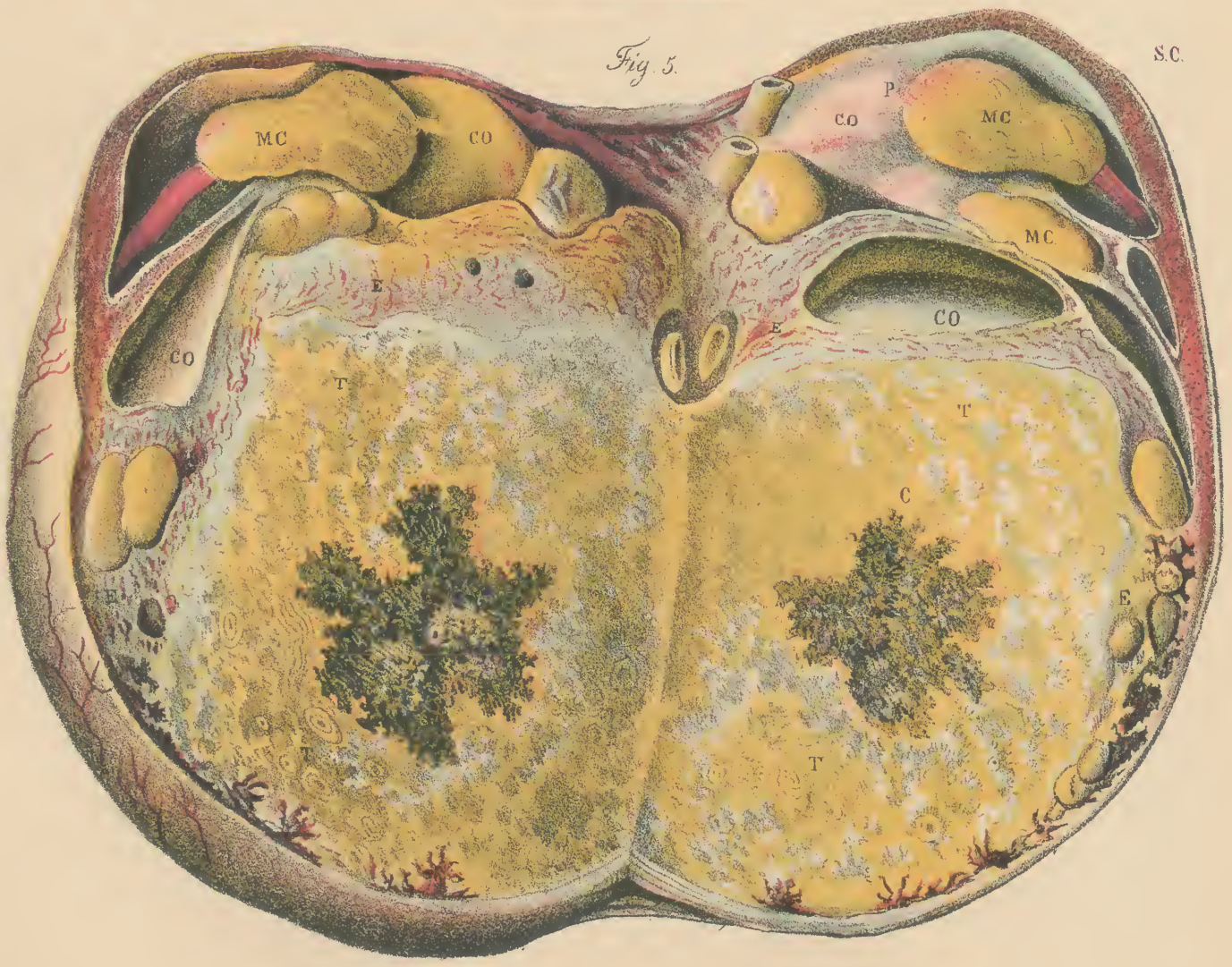

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TABLE IX.

FIGS. 1, 2—*Chronic Inflammation of the Right Kidney and Ureter.*

Case—A woman of 40 years, admitted into *Hospital la Charite*, with the following symptoms: irrepressible vomiting, very cold extremities, and state of depression of the nervous system. Died the day after admission. *Autopsy*—Uterus nearly completely destroyed; the same way the upper vaginal walls sloughed away by gangrene; subperitoneal abscess facing the anterior wall of the rectum, filling the Douglasian space; a large vagino-vesical sinus, passing into the fundus of the bladder; the tissues are black and sphacelous; the right ureter (*U D*) is enlarged, and its walls thickened; the mucous membrane of the opened passage (Fig. 2, *U O*) shows longitudinal thick folds, which form a kind of diaphragm, partly closing up the cavity; the right kidney (*R D*) is surrounded by a layer of fat (*T A*), very dense and intimately united with the proper tunic of the kidney, which is itself adherent to the organ, and could not be separated from it without tearing the tissues (*F R*) of the surface. The latter presented an embossed appearance, of a yellowish color and very dense. FIG. 2 represents the kidney opened and exposing the interior. The tissues are infiltrated; both the medullary and cortical substances are alike affected; the calyces (*P C*) are filled with fibrino-adipous neoplasma; the supra-renal capsule (*C S*, Fig. 1) is remarkably developed. (A section of it is seen in *Fig.* 2, *C C S.*)

FIGS. 3, 3¹, 4, 5—*Cancer of the Kidney and Ureter, forming a Renal Tumor.*

The kidney is laid open by a cut made from its convex to its concave borders. The interior shows a number of cavities containing serous liquid; almost the whole tumor (*T T*) is of a white semi-transparent, areolar structure, and containing yellow grumous matter; some calyces are transformed into wide pockets (*C O*), and contain carcinomatous substance (*U C*); portions of the cortical substance (*S C*) are structureless and granular; the pelvis is nearly obliterated; (*E*), encephaloid structure; the ureter (Figs. 3, 3¹) originated in the anterior superior part of the tumor; the pelvis (*B*) and ureter (*U*) are covered with enormous numbers of vessels; the cavities of both are filled with pultaceous matter (*C M*), similar to that contained in the calyces; (Fig. 4), magnified carcinomatous tissue.

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normal proportion; but the colorless gradually increase, and the colored decrease as the disease progresses

In old cases, a great quantity of excrementitious matter has been found in the blood by *Schotten; Picard* found four or five times as much urea in the blood of such persons as in that of healthy ones. This hypoglobulia explains the paleness of such affected individuals. The function of perspiration is nearly obliterated, and only returns when improvement takes place. In the very fewest of cases is there absence of dropsical effusion in some part or other of the body. As a rule, it is found either as local infiltration, or diffused all over the body from the beginning; in the acute form, the latter usually takes place, and the swelling is at once associated with the fever; in the chronic form they become gradually anasarcous, the face and hands being first affected with it. Not only the sub-cutaneous but the serous structure may become infiltrated, and the effusion may spread into the parenchymatous organs in the bodily cavities. The serous infiltrates contain much albumen and excrementitious matter (urea, etc.), which are eliminated from the body and temporarily prevent uræmic poisoning, but which eventually produce other no less serious consequences. The pathology of renal dropsy is as yet not quite explicit. The most feasible explanation is that, as *Frerrichs* stated, as the blood vessels dilate there is transudation into the tissues, and no absorption of the transudate takes place. *Jaccoud* considers the cause to be a modified condition of the blood, and consequently subject to chemical and mechanical changes in relation to the tissues and the vessels themselves. The most frequent complications and morbid accessory phenomena of this disease are *retinitis*, *gastro-enteric*, and *bronchial troubles*. Very rarely in the acute form, retinitis forms a constant and very valuable symptom in chronic Bright's disease. The trouble of vision develops slowly; the patient sees objects as if through a veil; he complains of spots upon the field of vision; soon amblyopia becomes more pronounced, and he is barely able to distinguish day from night. The progress of this retinitis is sometimes very slow, yet often a sudden attack of blindness may happen, and then be followed by some improvement; the eye-trouble is due to retinal hæmorrhages, which may be completely absorbed.

Examination with the ophthalmoscope will show whitish opaline spots around the papillæ (which may be sound); the spots may at first be separate, but they confluate and eventually form a ring around it. *Poncet* has proven that they are colloid and fatty changes of the fibers of the optic nerve; they become sometimes indurated or hypertrophic, prior to their fatty state; small hæmorrhages will be noticed as black spots upon the retina, and are often visible in a very early stage of the disease. *Brightic retinitis* may affect the nerve elements, or the connective tissue structures; the vessels may be sound or may present alterations, such as varicess, indurations, or fatty changes (*Virchow, Müller, Wagner, Schweigger*). According to *Gower*, retinal arteriitis in Bright's disease may be recognized by the reduced caliber of the vessels; such troubles are often curable.

Gastro-enteric and bronchial derangements form the commonest complications in Bright's disease. Dyspepsia, vomiting of glairy mucus, catarrhal diarrhœa, with pain in the bowels; sometimes even dysentery may co-exist; serous discharges in the disease are marked by their acid reaction (*Treitz*), or reversely, highly alkaline character and containing much ammonia; they may even sometimes contain albumen. In acute cases, diarrhœa seldom exists, except in the second stage; in chronic cases it is common; bronchial trouble never develops until the later stages of the disease, and is usually incurable; compensatory hypertrophy of the left ventricle is commonly produced in chronic nephritis; according to *Bright*, *Bamberger*, and *Rosenstein*, even in the second stage of acute nephritis, or in large white kidney.

Interstitial Nephritis (*Small Red Kidney*).

The first stages of this form are even more insidious and latent than the *catarrhal epithelial*. In cases consecutive to cardio-arterial lesions, the symptoms which first alarm the patient are: severe palpitation of the heart, great respiratory oppression, dizziness, and general debility. The pulse will show an increase of arterial tension, and the heart will manifest well-pronounced hypertrophy of the ventricles; a very frequent desire to pass water will awaken the patient from his sleep, and give him restless nights. Examination of the urine will at once reveal the disease. In cases uncomplicated with arterio-selerosis, the primordial symptoms are even more obscure than in the former. Very distressing dyspepsia, occasional severe headache, general debility, connected with strangury, may awaken suspicion of the existence of kidney trouble; sometimes even these indefinite symptoms do not exist, and the affected person becomes aware of his dangerous condition too late. The most reliable symptom in the early stage of this disease is *polyuria;* the quantity of urine may increase fourfold, but its density is not higher than 1,015, sometimes only 1,003, of slightly acid reaction, very seldom neutral or alkaline; at first it may contain a few blood corpuscles, afterward it leaves a very slight sediment, consisting of some debris of epithelium, blood cells, or hyaline cylinders. Albuminuria is not as copious as in the parenchymatous form. Dropsy sets in only in the later stages of the disease, and is neither general nor very abundant.

The hardness of the pulse, its breadth, and sudden rise of the wave distinguish this lesion, and differ from pulses in arterial lesions. *Bruit de galop* is characteristic of the heart-sounds; at its base there are three instead of two; an area of dull sound larger than usual shows hypertrophy of the heart to exist in this disease. *Potain*, who has thoroughly studied the causes of *bruit de galop*, ascribes it to the hypertrophic state of the walls of the heart; the thickened ventricle dilates slowly, and the diastole ends only after the contraction of its respective auricle; the third sound is then produced by the last act of the diastole of the ventricle. *Johnson* considers the third sound to be produced by the strong contraction of the now-enlarged and thickened auricle. Ocular symptoms exist in both forms of nephritis; cerebral hæmorrhages are more frequent in interstitial nephritis than in other forms.

Amyloid degeneration of the kidney is attributed to pulmonary tuberculosis, to long-continued suppuration in the body, and to syphilis; alcoholism, chronic articular rheumatism, and miasmatic fevers may also be predisposing causes of this form of tissue degeneration. Of late, the literature on this subject has become quite voluminous; a few authors may be mentioned: *Todd*, *Traube*, *Wagner*, *Tomaszewsky*, ——*Steward*, *Braun*, *Kuhne*, *Münze*, *Fischer*, *Fehl*, *Beer*, *Litten*, *Wolf*, etc., etc. As in other parenchymatous organs the degeneration takes place in the smaller arteries and capillaries, affecting chiefly the inner and middle coats, it spreads to the proper membrane of the tubules, and even the epithelium; the kidney becomes hard and hypertrophic, of lardaceous consistence, and of a yellowish white color; the cortical substance increases in volume, and readily separates from the other structures; its outer surface is either smooth or granular; a cut surface is lustrous like mother-of-pearl, and upon a yellowish ground shine a number of drops, which are degenerated glomeruli (*Meckel*). Amyloid kidney may be complicated with other degenerative forms of the tissues. *Symptoms:* Uncomplicated, it is essentially of a chronic nature; causes no pain in the kidney; in many cases, increased urination is often the only primordial symptom worth mention; it may eventually turn into polyuria; the urine is then pale, of very slight density (1,005–1,015 specific gravity); it deposits a slender sediment; the quantity of urea is diminished. The quantity of albumen in the urine is very variable. Dropsy seldom exists in this disease, and when it does set in it is only very late in its course (*Litten*), and is only partial, and develops slowly. *Retinitis*, cardiac-hypertrophy, and serous phlegmasiæ are very rare in uncomplicated cases of this kind; on the other hand, diarrhœa forms the most frequent symptom (*Meckel* and *Iuchmann*); hæmorrhages and hæmoptysis occur very frequently.

Suppurative Nephritis.

Compared with other renal affections, this is a rather rare disease. Yet not only are formations of abscesses and long-continued suppurations produced by mechanical injuries of the kidneys, but also by inflammation of the lower urinary organs (the ureter, the bladder) urethra, caused by retention of urine in those cavities for some time, inflammation in the perirenal structures; by general putrid infections, by metastatic purulent propagation. Formation of abscesses, with subsequent suppuration, is the rarest form of nephritis. Either one or both kidneys may become thus affected, according to the inciting cause being only on one or both sides. In acute suppurative inflammation the fever is intense, the lumbar pain very severe, more or less frequent vomiting, and hæmaturia; pressure increases the pain, movements of the body do the same, and extend it to the other urinary organs and into the testicle, which is drawn upward to the inguinal ring. In one-sided lesions, the left is usually the affected side (*Jaccoud*). The increase in volume of the organ is not always alike, and it may be ascertained by percussion (*Piory*) or by palpation (*Rayer*). The urine is the most characteristic phenomenon in this disease; the secretion is much diminished from compression of the tubules and glomeruli by exudates. As the inflammatory irritation extends to the ureters

TABLE X.

CYSTITIS AND PROSTATIC ENLARGEMENT.

FIGS. 1, 1¹ represent a very frequent lesion of the prostate gland, known since Sir Edward Home's description of it by the name of "*Development of the third lobe of the prostate.*"

The lobulated tumor (L M, Fig. 1) protruding into the bladder, made the discharge of urine from the cavity very difficult. The hypertrophy of that organ, indicated by its very thick walls, shows that it required strong effort to empty its contents. (C C C), folds of the thickened walls; (U U), vesical orifices of the ureters. The prostate is not only enlarged, but its tissues have undergone a kind of granular change; the verumontanum is bifurcated (U M) in front of the prostate.

FIG. 1¹—Longitudinal section of the bladder, showing the thickness of its walls; (C), a deep pocket, formed by a fold in its walls; (C P), section of the prostate.

FIG. 2—Ecchymosis in the bladder of a child, who died of hæmorrhagic small pox. The child was 5 years old. It died in the eruptive stage. The whole body, the parenchymatous structures, the skin, and the mucous membranes were filled with hæmorrhagic spots; the bladder presented both mucous and sub-mucous hæmorrhages.

FIG. 3—*Prostatic Calculus.*

It often happens that small calculi are lodged in the numerous orifices through which the prostate discharges its liquid into the urethral canal. In this figure (3) the posterior wall of the bladder is vertically divided, whilst the prostate is cut horizontally. A great number of brownish-yellow, small calculi are lodged in the prostatic cells (c p), the glandular structures of which are completely obliterated; the calculi were facetted as (C), a magnified grain, shows.

FIG. 4—*Vesical Phlebitis.*

The patient was affected with a lesion of the coxo-femoral articulation. The head of the femur was obliterated, the three pieces of the coxal bone separated, and caries existed in the iliac part of the cotyloid cavity; the veins surrounding the bladder were filled with pus, their perivascular tissues very dense, yet brittle; a quantity of matter was pressed out from the prostate (P), and seemed to have originated in the prostatic veins; the left common iliac vein (V P) and the orifice of the external iliac (same side) (O V I E) were filled with grumous blood, and part of the vesical veins contained both pus and clotted blood (P P).

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and to the bladder, tenesmus and strangury are produced, and with all efforts and straining the patient will discharge but a very small quantity of acid, scalding urine; in the further progress of this disease, this liquid will be of alkaline reaction, for a portion is generally retained in the ulcerated ureters or bladder, where this chemical change takes place; the density is generally normal, but now and then greater than usual. *Rosenstein* found the urates to be less than in ordinary febrile diseases. As a rule, the urine is concentrated and not albuminous, unless the case is complicated with other forms of nephritis, or in wounds of the kidney; hæmaturia exists then in the latter case. According to its cause, the disease may terminate in recovery, in death from uræmia or pyæmia, or may assume some form of chronic atrophy of the organ.

Chronic suppurative nephritis may exist a long time, unnoticed by the patient, so little are its symptoms pronounced. Pain in the kidney is produced only by pressure, when it will extend to the other urinary organs, and will produce temporary weakness of the lower extremities; the urine, in normal quantities, is of lesser gravity than the normal, slightly acid, and only in complications becomes alkaline; it contains mucus and pus, which fall to the bottom of the vessels after standing a while; the abundant white sediment thus found consists mainly of ammonia-phosphate of magnesia, and some albumen, derived from the pus and mucus. As a rule this disease ends in death. Hectic fever, at first intermittent, gradually becomes constant; very grave gastro-enteric derangements and general debility soon follow; *ammonæmia*, uræmia, or very fatal cystitis may intercurrently soon destroy the patient's life. Discharge of the abscesses into the tissues of the usually much-enlarged kidney, or into the adjoining tissues, are as fatal as the usual pyæmia, which, as a rule, ends his life (*Rayer, Sporer*). *Taylor* and *Stilling* have found portions of the kidney tissue swept out of the body with the purulent discharges from these abscesses.

Perinephritis and Pyelo-Nephritis.

The mucous membrane of the renal pelvis, like that of the biliary passages, may become affected with different forms of inflammation. *Membraneous pyelitis*, at one time *diphtheritic*, at others croupous, is a secondary lesion in infectious diseases and in cholera. *Catarrhal pyelitis* is brought about by gravel or urinary calculi, or by extension of inflammatory processes going on in the urethra, such as blenorrhagia, retention of urine in that passage, or poisons, cantharides, cubebs, etc. (*Schutz, Deutsche Klinik*, 1860. *Oppolzer, Wiener Spitalsz.*, 1864. *Hassall, London Lancet*, 1864. *Mall, Wien. Med. Zeit.*, 1866. *Philipson, Brit. Med. Jour.*, 1866. *Koster, Pyelo-Nephritis, Nederl. Arch.*, 1867. *Spencor Wells, Dub. Quar. Jour.*, 1867. *Filleau essai, Thése de Paris*, 1868. *Michaeli's Wiener Med. Presse*, 1870. *Dickinson, Transact. of Path. Society*, 1871. *Olivier, Arch. de Physiol.*, 1873. *Pascalluci, Il Morgagni*, 1873. *Finlayson, Glasgow Med. Jour.*, 1875. *Dauphin, Lithiase Renale, Press Med.*, 1875. *Pauli, Deutsche Med. Wochenbl.*, 1876. *Duncan, Clin. Lectures, Med. Times and Gazette*, 1878. *Ebstein, Deutsche Arch. für Med. Klin.*, 1878. *Ulzman, Wiener Woch. Presse*, 1880, etc.)

Pathological Anatomy.

In the lightest acute forms of catarrhal pyelitis there are only hyperæmia, hypersecretions of mucus, and epithelial exfoliation in the pelvis. In severe forms, the mucous membrane is injected and thickened (both of the pelvis and the calyces), and is covered with muco-pus; hæmorrhagic spots exist here and there, and the submucous tissue is infiltrated. In the chronic form, which is the most frequent, this mucous membrane is thick and of a livid color, or whitish, from the presence of phosphates in the pus. Ulceration of the surface is not uncommon, produced either by abraiding calculi or by corrosive exudates; the purulent liquid contained in the pelvic cavity has not the ordinary look of pus, but is viscid and jelly-like, from the action of the ammonia contained in the urine upon it. The semi-liquid nature of the contents forms already a sufficient obstacle to the free egress of the urine, but there are nearly always some solid substances (calculi, echinococci, phosphatic, and other concrements of solid pus, or similar emboli) to stop the flow into the ureters. Whatever the nature of the obstacle, there are formed dilatations and pockets filled with urine, pus, and sometimes blood. The pressure constantly exerted upon the tissues by these liquids is sufficient to produce atrophy, and it is then found that there is but a remnant of indurated structure, forming band-like walls around the dilated tumors. (Sec. VIII, Tabs. V, VI, VII, IX.) The volume of such tumors or pockets is sometimes astonishing; *Lebert* has found some to surpass that of the head of a grown person. When such a lesion is unilateral, the whole organ may become obliterated; by becoming atrophic and encased in connective tissue, be separated from all structures and thus rendered harmless. At other times the corrosive action of the urine and decomposible material will go on, and cause perforation through the structures, and empty the whole poisonous mass into the peritoneal or other cavities, causing death in a variety of ways. Under favorable circumstances a fistulous opening may be formed upon the surface of the body, or within a wide internal cavity, the contents gradually be emptied, and afterward the wound may heal by granulation. Such favorable terminations are generally very rare. Pyelitis is often complicated with abscess of the kidney (pyelo-nephritis). (Sec. VIII, Tab. VII, Figs. 1, 3, 6.)

Symptoms of Pyelitis.

If the origin of this inflammation is due to calculus, nephritic colics will exist in very painful paroxysms. Otherwise an acute attack of this disease will have the same symptoms, nearly, as acute nephritis, that is, there may be fever, pains, vomiting, and bloody urine. These symptoms will last a few days and then the disease assumes a chronic form, that is, the only valuable symptoms will consist in the anomalies of urine. The earliest alteration is the great increase of its quantity to an extent as to mistake the disease for *Diabetis insipidus* (*Oppolzer*). When the anatomical changes are great the quantity of the urine is lessened, its density is also reduced, the reaction is either acid or neutral, alkaline only when there is retention. Blood and pus, mucus in a floccular form contained in the urine will form a whitish or brownish sediment in the vessel. The jelly-like substance formed by the ammonia shows its pus-like character, and when epithelial cells, which are always present in great quantities, are of the imbricated kind, the microscope will at once show the disease to be that of the mucous membrane of the pelvis. The quantity of albumen is proportional to the quantity of pus. The presence of much pus in the urine renders it milky and the two only separate on standing. This is characteristic of pyelitis; in accidental admixture of pus to urine the two liquids are not intimately mixed. Large quantities of phosphates are, as a rule, also contained in pyelitic urine. The passage of water is generally free in this disease, and becomes painful only in acute exacerbations, which are of frequent occurrences. In the calculous form of pyelitis there are two particular symptoms characterizing it; one is, that besides the ordinary fixed pain of the kidney trouble there are paroxysmal attacks of colic, at which time the urine becomes temporarily bloody. Secondly, that the character of the urine is very variable; that after an attack of colic the urine may become perfectly normal, as *Richardson* has found the ureter closed up on the diseased side. When such is the case for any length of time it may be presumed that the person is cured. In bilateral pyelitis the closure may take place on both sides and the patient then dies of anuria. Occlusion of the ureter, even in unilateral pyelitis, is not always without danger, as *Bourgeois* has observed in several cases, when retention in the one ureter produced anuria and death. Hectic fevers and general marasmus often follow very extensive pyelitis. Disturbances of digestion are very common in this disease. When the pyelitis progresses slowly a lumbar tumor is formed with renal atrophy and the patient may survive it a long time. The character of the urine will be but little changed, and the disease may then barely be suspected. Occasionally such tumors may be mistaken for hepatic, splenic, or other abdominal tumors. However, as tumors of other abdominal viscera are seldom situated in the renal region, and the movements of the diaphragm are without effect upon renal tumors whilst they displace hepatico-splenic enlargements, differential diagnosis need not be so very difficult. Complicated with perinephritis, there will be enormous pains in the renal region which extend to the regions of the crural and sacral plexuses, and will be connected with violent fevers. There is often contraction of the psoas muscle. When a tumor is once formed the urine may or may not contain pus, according as the ureter is open or closed. Such tumor may break into the tissues, then discharge its contents and produce fatal consequences, or open externally with very variable terminations.

Renal Lithiasis and Nephritic Colic.

Renal concretions may deposit as sand or gravel in the papillæ or tubules, or as more voluminous masses, occupying the papillæ, the calyces, mostly the renal pelvis. The first constitute the so-called uric

CYSTITIS,

PROSTATIC ENLARGEMENT.

Sec. VIII. *Tab. X.*

Fig. 1.

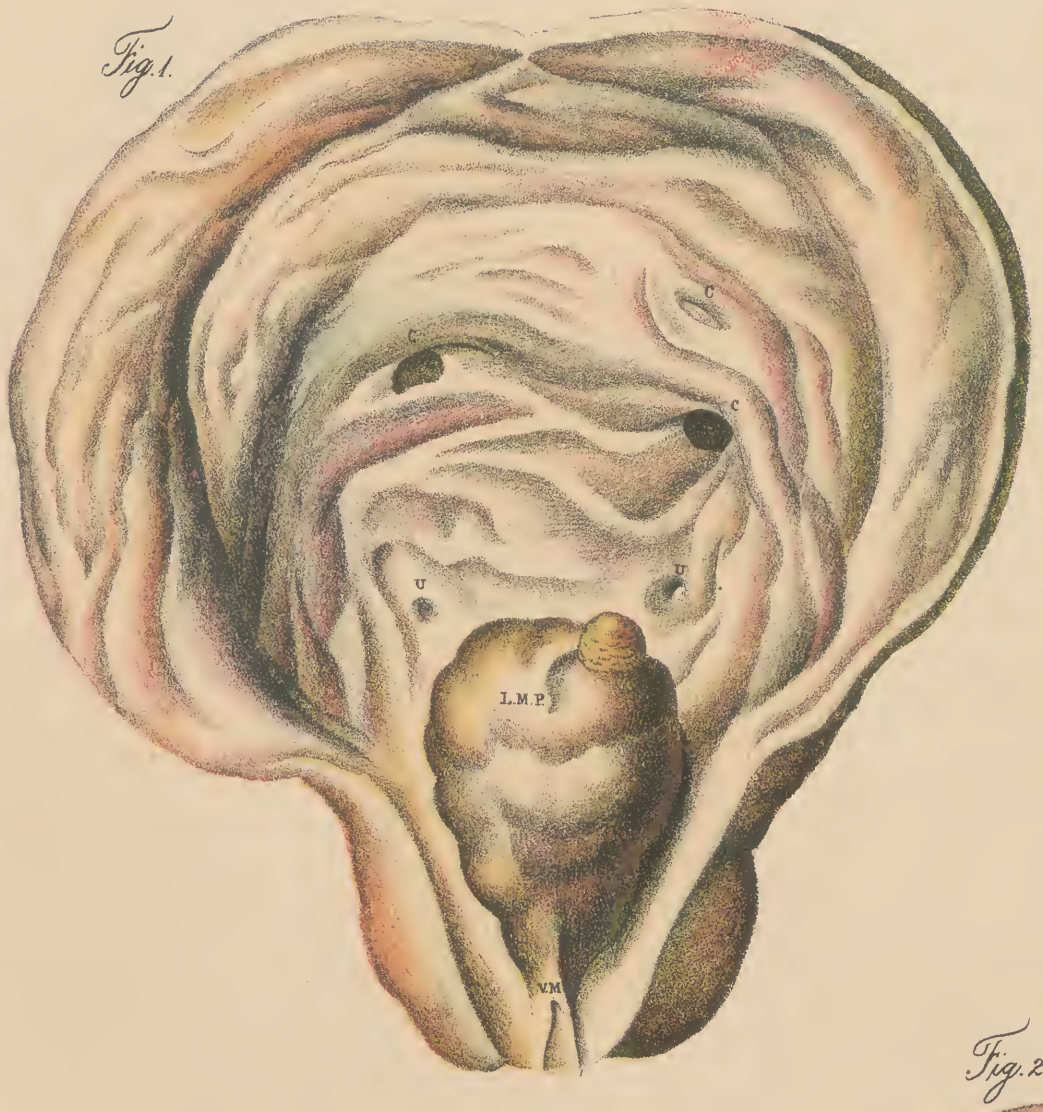

Fig. 1'

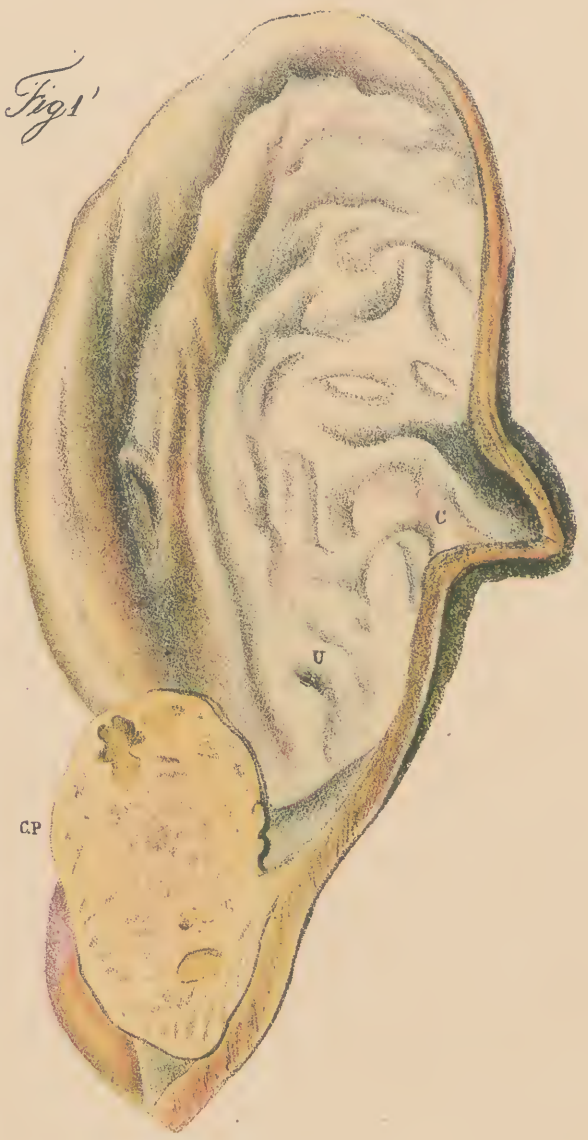

Fig. 2.

Fig. 3

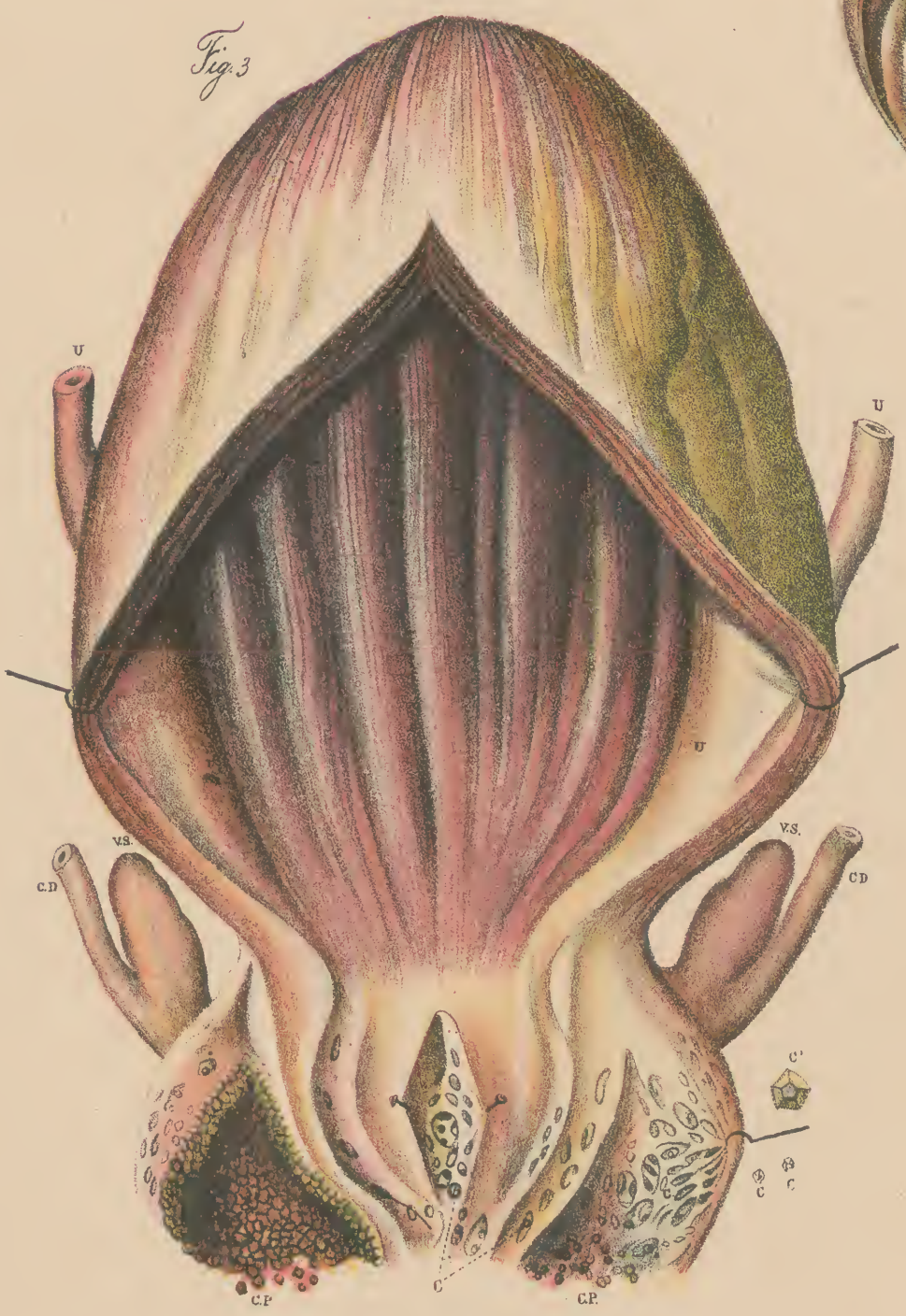

Fig. 4.

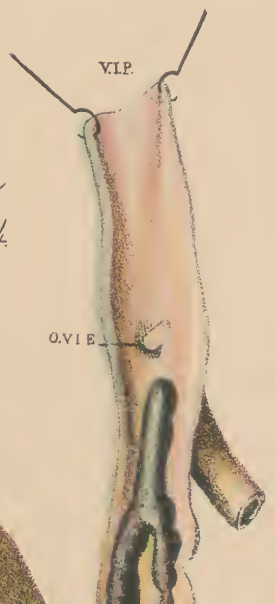

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Fig. 1.

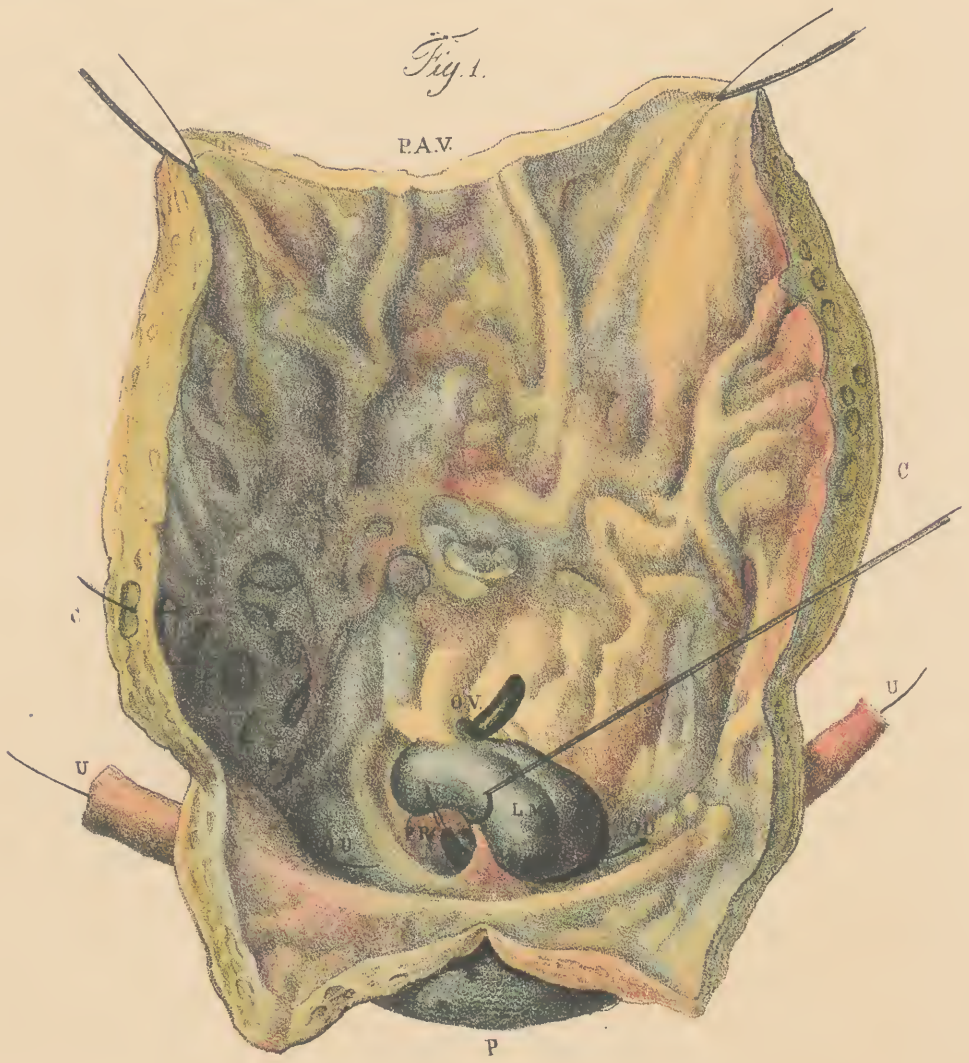
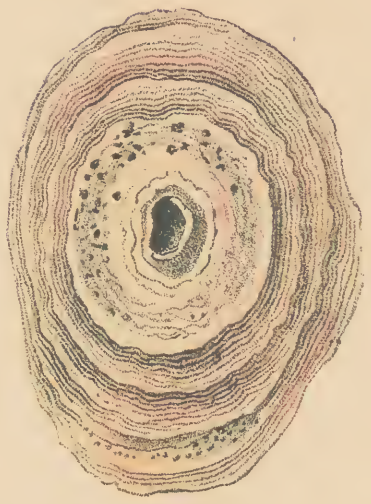


Fig. 6



6'

Fig. 5.

Fig. 2.

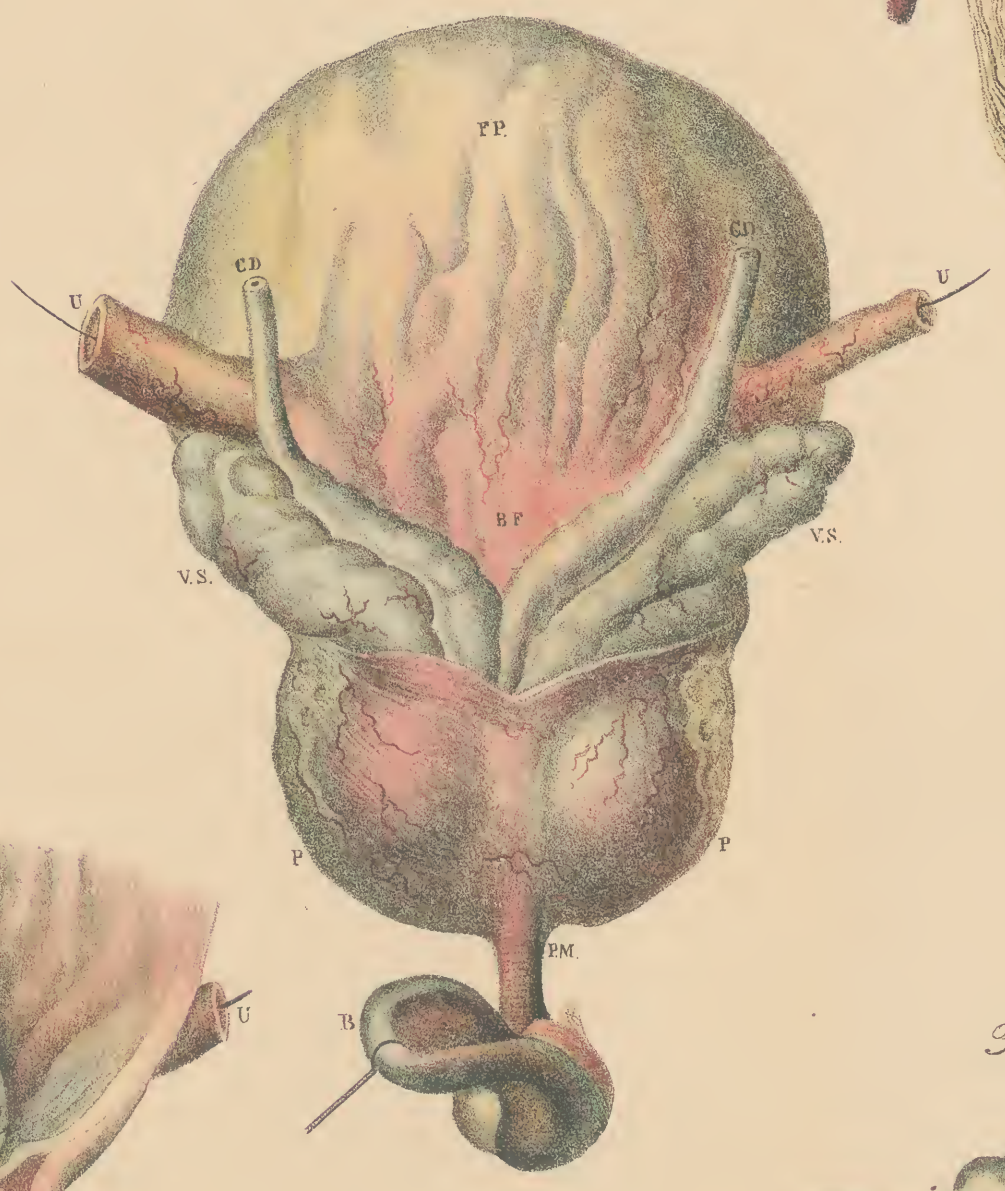


Fig. 3

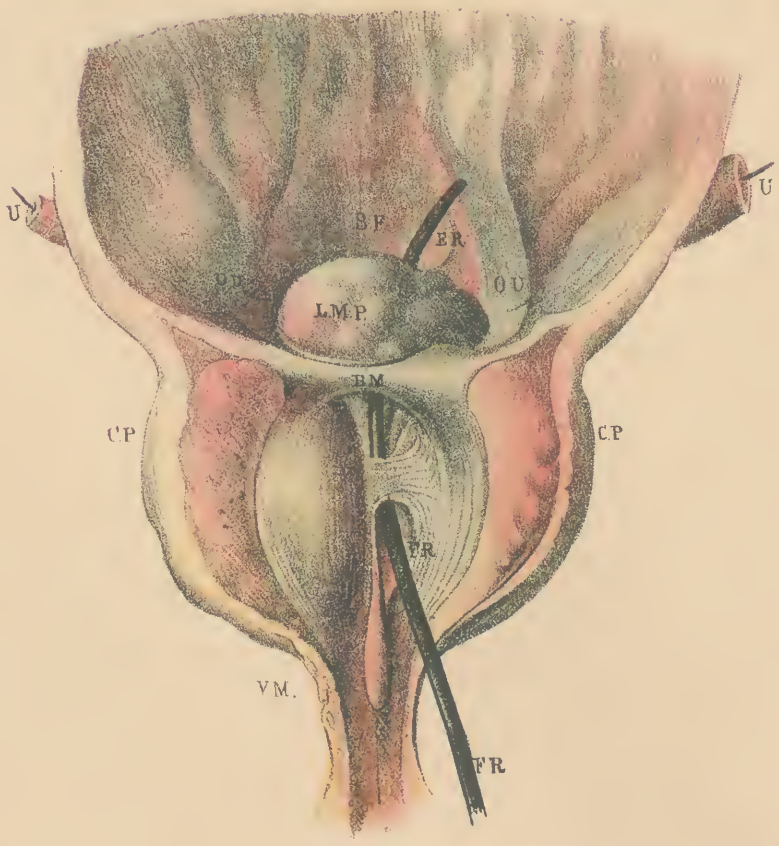


Fig. 4.

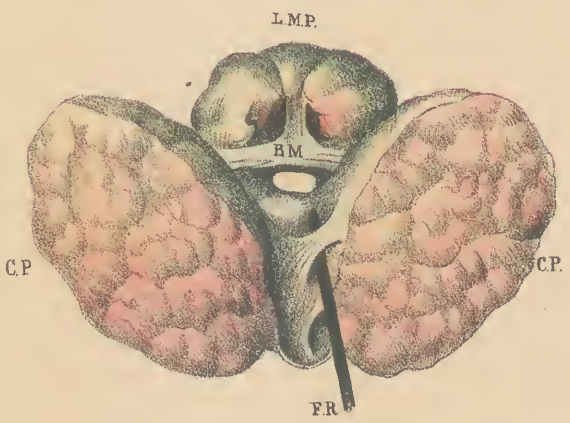


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TABLE XI.

FIGS. 1, 2, 3, 4, 5, 6, 6[1]—*Cystitis. A Tumor and a large Uric-Acid Calculus in the Bladder.*

Case—A man about 40 years. Had suffered for years from chronic cystitis, producing constant desire to micturate, with frequent retention. A violent attack of pains and obstinate retention was temporarily relieved by catheterization at first, and afterward by production of an artificial canal in the mucous membrane of the urethra. Seemed to improve for about six months, when a new attack of acute cystitis, with obstinate retention, high fever, and inflammation, produced pyæmia and shortly afterward death. In the post-mortem examination a very large calculus was found in the bladder.

FIG. 1 represents the posterior wall of the bladder divided, showing the anterior wall to have a bluish-gray color (P A V), common in chronic cystitis. It is very thick and hypertrophied, presenting broad folds in every direction. A number of pockets are formed in the walls, and a number of cysts project into the cavity of the organ (C C); a narrow orifice of those cysts terminates in the cavity also; (U U), dilated ureters, through which bristles are passed, terminating in the vesical orifices of those canals (O U, O U); (L M), a tumor, is seen at the bottom of the anterior wall projecting into the bladder; above it is the vesical opening into the urethra (O V); below is the opening of the artificial channel made by the operation (F R); (P), thickened prostate.

FIG. 2—Posterior aspect of the bladder. (F P), Vesical portions of the ureters (U U); (V D), *vasa deferentia;* (V S), vesiculæ seminales. The prostate (P P) presents its inferior surface; it is very large and covered with many dilated vessels; a quantity of small calcareous granules are lodged within its tissues. The membraneous portion of the urethra (P M) and the bulb (B) are very much enlarged and thickened.

FIG. 3—The lower portion of the anterior wall of the bladder, the upper wall of the urethral canal, and the prostatic wall are vertically divided, exposing their cavities. (B F), the posterior inferior cul de sac; (L M P), prostatic tumor; (O U), urethral orifice; (C P), prostatic portion of the urethral canal; (V M), verumontanum; portion of neoplasm closing up the natural urethral opening, (B M); (F R), border of newly-formed vesical opening. A sound indicates the newly-formed canal.

FIG. 4—Perpendicular cut of the prostate (C P); (L M P), prostatic tumor; (F R), portion of the wall of newly-formed passage, and sound passed through it; (B M), membraneous wall of the orifice attached to the tumor.

FIG. 5—External surface of uric-acid calculus.

FIG. 6—A section of the longest diameter of the calculus, showing its concentric layers and a clot in its center (6[1]).

infarcts. The second are the calculi, which descend from the kidneys into the ureters and the bladder, and increase in size as they descend.

Renal lithiasis affects old and young, but is more frequent in old age than at any other period of life, and more common among men than in women. *Jaccoud* (*pathologie interne*) finds that uric-acid calculus exists in equal frequency in both sexes. In some countries it exists more frequently than in others. The reason of its unequal frequency is as yet no better known than the probable cause of its production. There are two theories in regard to its production; one is, that there exists some peculiar constitutional disposition (diathesis) to produce an excess of the composing elements of the gravel or the calculi; the other is, that it is formed by a lithogenic catarrh (*Meckel*) in the kidneys, and is of local origin. At present the prevailing opinion is, that it is the product of certain kinds of fermentation going on in the kidneys. There are certain facts favorable to the first theory, that is, the well-ascertained facts as to its hereditary transmissibility, which certainly looks more like a general peculiarity of the circulation and nutrition than otherwise.

Like biliary calculi, the urinary may be latent for quite a long time, and then call forth dangerous pyelitis or hydronephrosis. They may also be driven from the body with but little inconvenience to the affected person, or with very severe colic-like pains (nephritic colic). Attacks of the kind may be preceded by dull pain and sense of pressure in the lumbar region for weeks or months, with considerable weakness in the lower extremities, or may come on all of a sudden, and surprise the apparently healthy person attacked. The pain, usually unilateral, is very violent, and spreads from the lumbar to the sacral and pelvic regions; besides the pain, the pulse is very small; the patient's face is covered with cold sweat and is pale, and the extremities are cold; urinary secretion is diminished and micturation very painful. A few drops, now clear, then turbid, bloody, or mixed with mucus, will slowly pass. The pain produces in very sensitive persons and young children very violent reflex-convulsions. The duration of such attacks is variable. After the attack the flow of urine is generally very free, *and may contain gravel or urinary calculi.*

Peri-Nephritis, Phlegmon.

Phlegmonous inflammation of the cellulo-adipous structures surrounding the kidney, although a rare primary lesion, occurs frequently as a complication in local and general diseases. The causes of the primary form are as yet not ascertained. The secondary inflammation may follow wounds, both mechanical and chemical injuries, violent exercise on horseback (*Turner*), or by severe jolting of vehicles (*Jaccoud*). Long and fatiguing marches, strains in lifting, etc., are the commonest causes of this disorder. Inflammation of the adjacent tissues, or those connected with the kidney physiologically, very often give rise to peri-nephritis, pyelitis, and pyelo-nephritis. Calculi, causing perforations and infiltration of urine into those tissues, even without perforation, causing the inflammatory process to spread into the tissues, and renal lithiasis may be ranked first as inciting causes of these phlegmonous inflammations. *Hydatic cysts* (*Rayer, Beraud, Denonville*), *strongelus giganteus* (*Chopart, Maublet, Lapeyre*), and *cancer of the kidney* (*Cornil*) may, under certain circumstances, produce this lesion; renal tuberculosis (*Rayer, Naudet*) often causes peri-nephritic phlegmon. Likewise, even distant phlegmasiæ may bring forth the same morbid process, such as cystitis (*Tachard*), hepatitis and inflammation of the biliary passages (*Millard*), phlegmasia of the psoas muscle (*Rosenstein*), pelvic cellulitis (*Chopart*), etc. *Duplay* has observed peri-nephritis during convalescence from typhoid fever; *Rosenstein*, in typhus exanthematicus; *Wagner*, in small pox; *Trousseau, Gueneau de Mussy*, in puerperal inflammations. As a primary or a secondary affection, when the cause is local, the disease is unilateral (*Jaccoud*); when from constitutional trouble, both sides may be affected (*Rosenstein*). *Nieden* found that in 166 cases, 97 were males. From the age of forty and upward the disease is the most frequent, yet *Duffin* and *Loeb* have found children from four to six years of age to suffer from it.

Primary phlegmonous peri-nephritis offers the most pronounced symptoms. It begins with a violent chill, and is soon followed by a very high fever, often followed by profuse perspiration. It assumes occasionally a distinct intermittent type, and thus leads to some errors in diagnosis. Generally quotidian, it may become tertian, or assume a remittent form, with nightly exacerbations. The tongue is *white* and *dry;* loss of appetite, great thirst, and obstinate constipation usually mark its character. Vomiting is rare, except when complicated with other nephritic derangements. Pain in the region of the inflammation is often the earliest, and then the most important symptom; it may be stinging or dull pain, constant or intermittent, or appear with the chill (*Lecorche*). *Jaccoud* and *Velpeau* have noticed that the pain spreads to the sacral and crural regions, and imitated *crural neuralgies.* On one hand paresis, there may be, on the other hand, contraction of the psoas muscle, and trouble of the lower limb on the same side. After a few weeks lancinating pains appear, with formation of a tumor in the lumbar region. The integument over the tumoral region presents a slightly œdematous swelling, at first diffuse, subsequently circumscribed, and becomes prominent, of a more or less red color. The more the abscess approaches the surface the more will it be recognized by superficial fluctuation. The pus does, in rare cases, penetrate the transverse muscle, spreads in the sub-cutaneous cellular tissue, raises the skin, and becomes perceptible as a superficial abscess. In primitive peri-nephritis, the suppurative process develops in about two weeks; in secondary it has no definite development, and depends on the progress of the primary disease; in primary forms the urine is nearly normal; in traumatic there is generally hæmaturia, albuminuria, etc. The suppuration has a variable course and termination. It may discharge into the region where the most lumbar hernia exists (*Jaccoud*), and then the fever and other severe symptoms will subside, or it may infiltrate in the different structures, and give rise to dangerous and complicated ailments, and terminate in death.

Opening of the abscess in the colon was considered, by the older physicians, to be a favorable issue, for the pus will soon produce a brisk discharge of the intestinal contents, and its own elimination will be affected (*Rayer, Cruveilhier, Chassaignac, Parmentier, Hervieux, Cornil, Naudet*). Discharge of the abscess into the peritoneum, fortunately rare, is generally the most dangerous accident to the patient; speedy death, or long, lingering complications take place. The course which the pus will follow is often most singular, for it may empty anywhere in the abdominal, thoracic, or pelvic cavities, produce secondary abscess or gangrene in any of the viscera lodged there. Even under the most favorable circumstances phlegmonous peri-nephritis must be considered one of the most dangerous, the least manageable, therapeutically or surgically, and often the most difficult to diagnose of diseases situated in the lumbar regions. Death is the frequent termination; recovery, even partial, is very rare.

Catarrhal Inflammation of the Bladder.

Cystitis may be produced by traumatic causes, such as all sorts of mechanical injuries, hard childbed, or from unskillful catheterization, etc.; infections from rectal or vaginal discharges, or irritation produced by poisons upon the mucous surfaces, or absorption from the blood, or the reaction caused by urine itself upon the walls of its cavities in different diseases, may provoke catarrh of the bladder. It is a common disease in very old age, and more frequent in men than in women. The causes alone determine whether the disease will become chronic, or remain in the acute form.

The inflammation is usually confined to the trigone and the base. When it is generally inflamed, it is more intensely affected at the neck. In acute inflammation, the mucous membrane is hyperæmic, swollen, and soft; the glands are swollen and injected, and the surface of the membrane is covered with more or less opaque mucus and debris of epithelial cells. In very severe cases there is fibrinous exudation, either superficial or interstitial; the latter give rise ulteriorly to erosions and alterations of the mucous membrane. In chronic cystitis, the color of the organ is brownish or grayish-blue (*Sec. VIII, Tab. XI, Figs.* 1, 2, 3, 4); the mucous structures, thick and knotty; the sub-mucous, connective, and muscular tissues are hypertrophic; the vesical cavity is generally diminished in size; the muscular bundles seem more voluminous than usual; the mucous membrane is often pressed back between muscular bands, and forms pockets of great size, which communicate with the cavity of the bladder by means of narrow orifices. These often become the seat of calculi and ulcerations. The internal surface of the organ is covered with puri-form mucus, or yellow pus, and urine contained in it is alkaline, and evolves an odor of ammonia. When the catarrhal process is produced by prostato-urethral alterations, or by a calculus, a number of complications and sequels will follow; these may be: mucous or sub-mucous alterations, which may cause perforation of the bladder.

When adhesions to the adjacent tissues are produced by

TABLE XII.

FIGS. 1, 1^1—*Tuberculous Degeneration of the Prostate.* This alteration spreads, on one hand, to the seminal vesicles and *vasa deferentia;* on the other, to the bottom of the bladder. The degeneration extends into the bladder beyond the *trigone;* behind, two bristles are placed to show the narrow prostatic orifices. FIG. 1—Posterior surface of bladder; (V S D), *vasa deferentia* and seminal vesicles; (C D), *vasa deferentia;* (U), ureters; (B u), bulb; (P), enlarged prostate. FIGS. 2, 2^1 represent the prostate and the bladder of a man of 81 years. He died of cancer of the stomach. FIG. 2^1 shows the bladder opened in front; it has undergone sclerosis of all coats. (V), bladder; (C D), *vasa deferentia;* (C P), the altered prostate, which forms a series of cells, filled with serum and pus. FIG. 2 shows the contracted urethral orifices (U U). FIGS. 3, 3^1, 3^{11}—An extraordinary congenital deformity. There were two urethral canals, one superior, smaller (Fig. 3^1), occupying the superior surface of the penis, in the median line; the other, inferior (Fig. 3^{11}), in its normal situation, and normally constructed. Both opened into the gland (Fig. 3^{111}), viz: the inferior, into the normal place of entrance (U U); the superior canal on the superior surface of the gland, into a small and narrow orifice (O C E) of the inferior canal. The upper canal was formed of a junction of two ejaculatory passages, at the root of the penis. (C B U), canal of the bulb; (C U O), normal urethral canal; (U U), *meatus urinarius;* (C E S), superior abnormal canal; (C E C E), ejaculatory passages.

peri-cystitis, the abscess may empty into the rectum, into the vagina, or even outside. The danger of urinary infiltration is thus prevented, but on the other hand the discharge may take place into the peritoneal folds, and dangerous peritonitis may follow; the pus may also corrode the membrane, and produce perforations, with infiltration of urine into its folds. There may also be formed thickening and sclerosis of the bladder, the muscular tissue becoming enormously hypertrophic, and diminish the cavity of the organ.

Symptoms.—Acute cystitis begins sometimes with a high fever, but, as a general thing, pain and derangement in urination and alteration of the urine are the prevalent phenomena. The pain may vary in intensity, may be only confined to the organ alone, or may spread to the perineum, the anus, the testicle, or even to the lower extremities. The pain of the bladder is, under all circumstances, most distressing; pressure upon the organ, bodily movement, or the contact of the urine with the mucous surface of the organ increases it. The intolerance of the inflamed membrane to the urine is the cause of the constant desire to urinate, and the terrible effort the patient makes to empty the organ. The neck of the bladder becomes painfully contracted after the expulsion of the urine. *Dysuria*, *ischuria*, and *tenesmus* exist nearly at the same time; under such circumstances, of course, introduction of any sound or other instrument is inadmissible. The quantity of urine is generally diminished; but the kidneys may still continue to secrete the normal quantity, and the cause is then *retention in the bladder*, which is often full, and excruciatingly painful. The urine at first contains but little mucous, but in a day or two afterward it contains great quantities, which form a whitish sediment. There is generally constipation of the bowels; but when the inflammation spreads to the rectum there is rectal dysentery. Acute cystitis may speedily subside, when its cause is transient, may last a long time, or pass into a chronic form. In old age the disposition is to assume a chronic course.

Chronic Cystitis.—Although less painful, and attended with less severe symptoms, it is yet the more dangerous of the two forms. In old age it becomes persistent, and induces a great variety of lesions, which destroy the patient's life. Painful micturation and dysuria very seldom exist; tenesmus, sometimes in the discharge of the last drops. The urine is generally pale, of little density, and abundant. At first slightly acid or neutral, fairly clear, it soon becomes turbid or opalescent, and the mucus becomes muco-pus. The sediment is cohesive, yellowish white, sometimes viscous and jelly-like; pus and epithelial cells of the bladder and crystals of phosphates are found in the deposit. The urine gradually becomes alkaline from the fermentation which it undergoes in the bladder, and smells strongly of ammonia. The course of chronic cystitis is very irregular, its duration uncertain, and its termination very variable. Persistent causes, of course, prolong its existence and render it incurable; sometimes many derangements and ailments are thus induced—gastric disturbances, enteric, and other troubles. Even nervous-central lesions take their origin in ammonæmia; or ulceration, with subsequent gangrene and septicæmia, often follow in its train. *Hypertrophic cystitis* often gives rise to very grave errors in the treatment and diagnosis. The muscular paresis produced thereby prevents complete emptying of its cavity; a portion of it remains and gives rise to fermentation, with subsequent ammonæmia, which generally carries the patient off. (*Purdon, Chronic Cystitis, Dublin Jour. Med. Sciences*, 1873. *Perrin, Thése de Paris*, 1874, etc. *Hannot, Catarrh Chronique, Presse Med.*, 1875. *Bell, Exfoliation, Edinburgh Med. Jour.*, 1875. *Dubelt, Arch. f. Exp. Pathol.*, 1876. *Edelfsen, Deutsch. Arch. f. Klin. Med.*, 1876. *Johnson, Clinical Lectures, Lancet*, 1876. *Duplay, Cystite Progres Med.*, 1877. *Girard, Thése de Paris*, 1877. *Hicks, London Lancet*, 1879. *Galliet, Gazette Hebdom.*, 1880. *Guyon, Leçon, Paris*, 1881, etc., etc.)

Uræmia.—The name of uræmia is given to divers morbid derangements, which have a common cause, that is, "insufficiency of urination" (LEPINE, *Additions to Bartel's Diseases of the Kidneys*). "I use these words," says this author, "instead of urinary retention, to distinguish them from those cases which are affected with *uræmic cachexia*, and present symptoms, as follows: respiratory derangements, vomiting, or diarrhœa, a moist tongue, convulsions, and coma; the others present typhoid symptoms, they seldom vomit, bowels are constipated, the tongue and other mucous surfaces parched, the intellect is clear, no dyspnœa." Cases of long retention of urine are cited by *Loring* (*Urinary Diseases*, 1838), *ten days' anuria; Rayer* (*Maladie des Reins*), *ten days' anuria; Picard* (*Gaz. Med., Strasburg*, 1879), *nine days; Paget* (*Transactions*), *fifteen days' anuria and death; Fuller* (*Transact., Vol. XIV*), *Bagshaw* (*Transact., XIV*), *Tenneson, Tournadre, Peschek, Mendel, Salgado, Schwengen, Anwell, Dubuc, Haechner, and many more authors*, have reported cases of retention of many days' standing; some recovered, some died.

Although essentially alike in the proximate cause, the distant causes may differ very materially. Sometimes there are more or less toxic substances in the blood (*potash, ptomain*—VOIT, FELZ, RITTER, ASTUSCHEWSKY), or an accumulation of such substances which act injuriously only mechanically, such as urea, etc., or œdematous exudations in one or the other of the parts of the nerve centers (*Bouchard, Lichtenstern, Mathieu*). Vascular derangements may also give rise to uræmia. It would be a very fortunate thing for the practitioner if, in some cases, he could lead the disease back to its real cause. So far the symptoms, however pronounced, fail to give positive references to their inciting, primary causes, except the one tangible *anuria*, or *long-standing* retention.

Diabetis Melitus, Glycosuria.—Unlike the above-named lesions, diabetis melitus is only a secondary affection of the kidneys, produced by the enormous labor of elimination from the body chiefly of the great excess of glucose contained therein; also from the local action produced upon the tissues of the kidney by the eliminated substances (sugar, creatin, uric acid, etc.), or by absorption into its tissues of those substances. There are secondary and primary symptoms, which must be separately noticed. The main symptoms characterizing the disease are: polyuria, glycosuria, polydipsia, polyphagia. The quantity of water discharged during the 24 hours may amount to 6 or 7 litres. Of course, a quantity equal to the one discharged will be necessary to replace it, and hence enormous *thirst* will be felt by the patient (polydipsia). Discharge of pale, acid urine may go on for a long time without the affected person becoming aware of his disease, until some unexpected circumstance reveals its nature. In the early stages, diabetic patients keep well up in flesh, sometimes even in vigor. Only the annoyance of having to get up from sleep to pass water, and an extra thirst, will sometimes awaken suspicion of there being *something wrong with the kidneys.* The quantity of sugar passed during the 24 hours may amount to 600, 700, even 750 grammes. This *glycosuria* is at first still the result of formation of enormous quantities of glucose from the starchy food and other carbo-hydrates introduced into the body. As long as this lasts, the body will not require more nourishment than usual. There will at first be much hunger, but no absolute craving for *great quantities of food. Polyphagia* will begin when part of the nitrogenous food taken becomes disassociated, and converted into glucose. Of course, as the disease progresses, and the quantity of formation and elimination of sugar goes on, and especially when sugar is formed not only of the foods but also of the patient's own tissues, then the desire for food—although sometimes co-existent with dyspepsia and even disgust for it—will become extraordinary. There will be *autophagia, self-consumption*, added as a symptom. The quantity of urea in 24 hours varies very much, yet it is never below the normal (*Jaccoud, Leçons Cliniques*). In diabetis melitus, glycosuria co-exists with polyuria and the other symptoms. There may be glycosuria alone, as in injuries of the brain and nervous system, or polyuria alone, as in the early stages of Bright's disease, but neither of these can be considered diabetis. In this disease there is glyceæmia, or sugar, in large quantities, in the blood, sufficient to produce polyuria.

Secondary Symptoms.—can, *a priori*, be expected to exist in a condition of the body when the blood is enormously altered and the functions so radically affected. The great quantity of glucose contained in the blood is eliminated through every excretory organ. The skin, by profuse perspiration, throws out of the body notable quantities of sugar (*Griesinger, Semmola*). *Vogel* found, in one case under his treatment, that the quantity of sugar eliminated by perspiration exceeded that emitted with the urine. *Zabel* found equal quantities of glucose in the fæces of diabetic patients as in the urine. Before meals the saliva is usually acid, and sometimes even afterward for some time. This is due to formation of lactic and butyric acids, by fermentation of sugar secreted with the saliva, brought about by microphytes, constantly present in the mouths of such patients. The teeth become carious and are loosened in their sockets, and either soon drop out or have to be extracted. Stomatitis is a very common complication in this disease. The peculiar disposition of diabetic patients to become affected with all sorts of phlegmasia is due to an anomalous state of the blood. *Anthrax* is one of the frequent forms of superficial phlegmasia. In Brazil, South America, where it is so common, *d'Aquino, Fonceco, Iardao*, and *Marchal* have made valuable observations on the subject, and many physicians in that country connect the two lesions as a matter of course. Erysipelas is another frequent secondary symptom. Pneumonia, a very frequent complication of the disease, often appears in the early stages, and often leads to pulmonary gangrene, which, according to *Moneret* and *Scot*, differs from ordinary pulmonic gangrene. Similar mortifications are observed in the extremities of such persons, which seem due to fermentation of the glucose. Very frequent urination gives rise to redness of the *meatus urinarius*, and to an insupportable itching. Prurigo and herpetic eruptions also exist, and may spread upon adjacent parts. Swelling of the prepuce, phimosis, and balanitis are common in the disease. Not only the distressing thirst, but also constipation, and dryness of the skin and mucous surfaces are due to the enormous losses of water from the body. Pulmonary phthisis, according to *Griesinger*, destroys forty-three per cent. of diabetic patients. *Graefe* has found very many cases of cataract in this lesion.

Fig. 1.

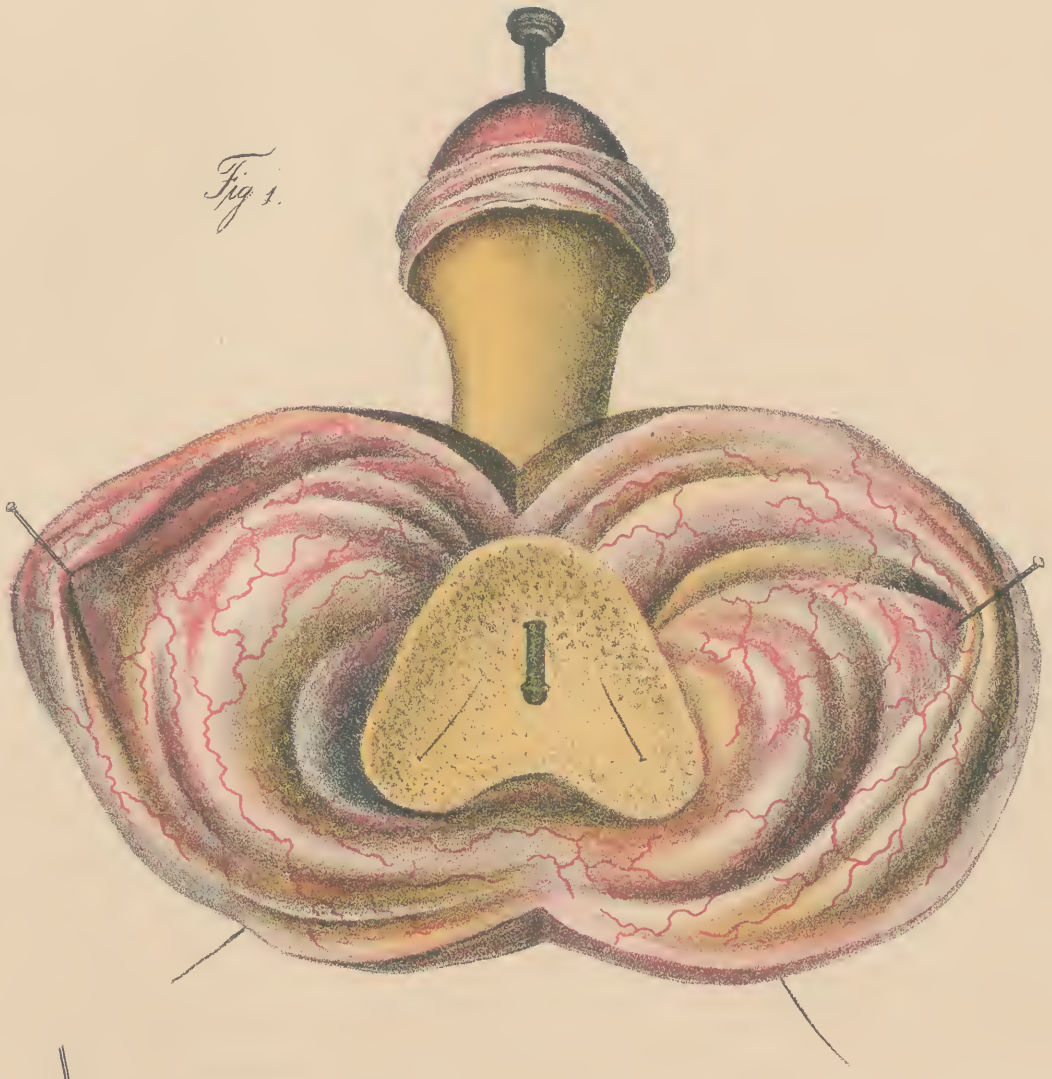
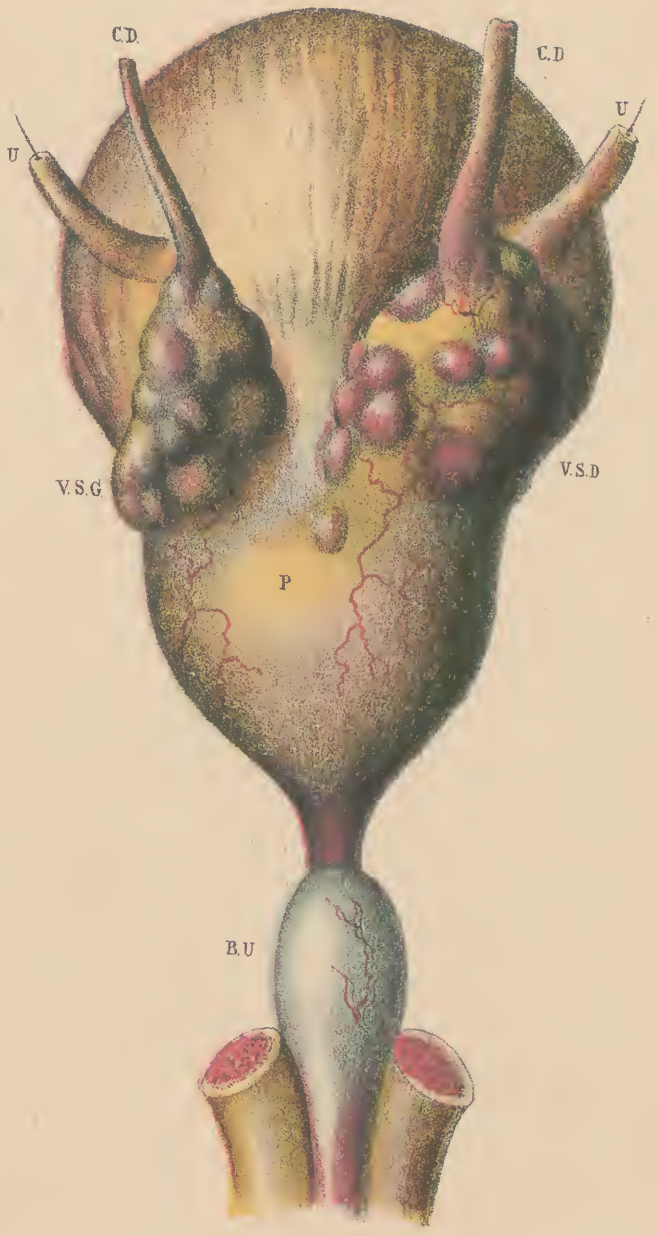


Fig. 1'



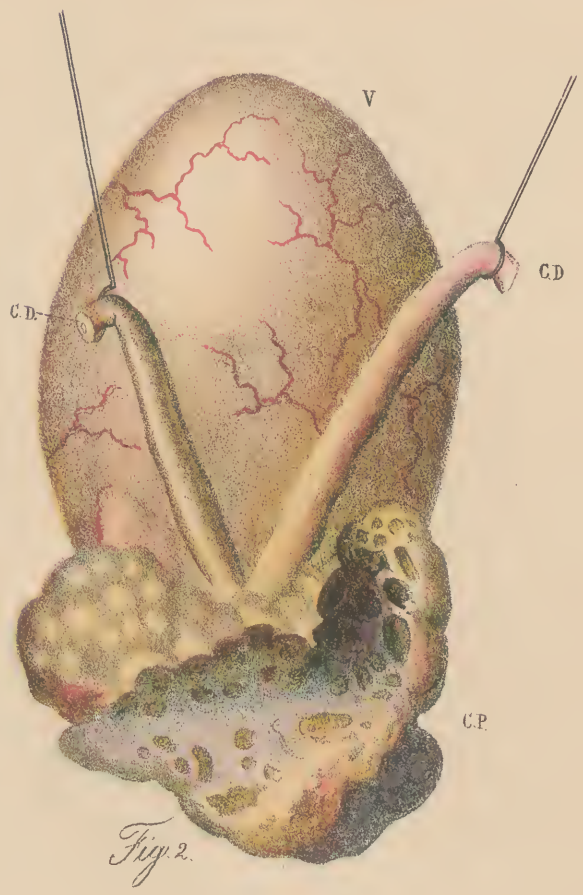


Fig. 2.

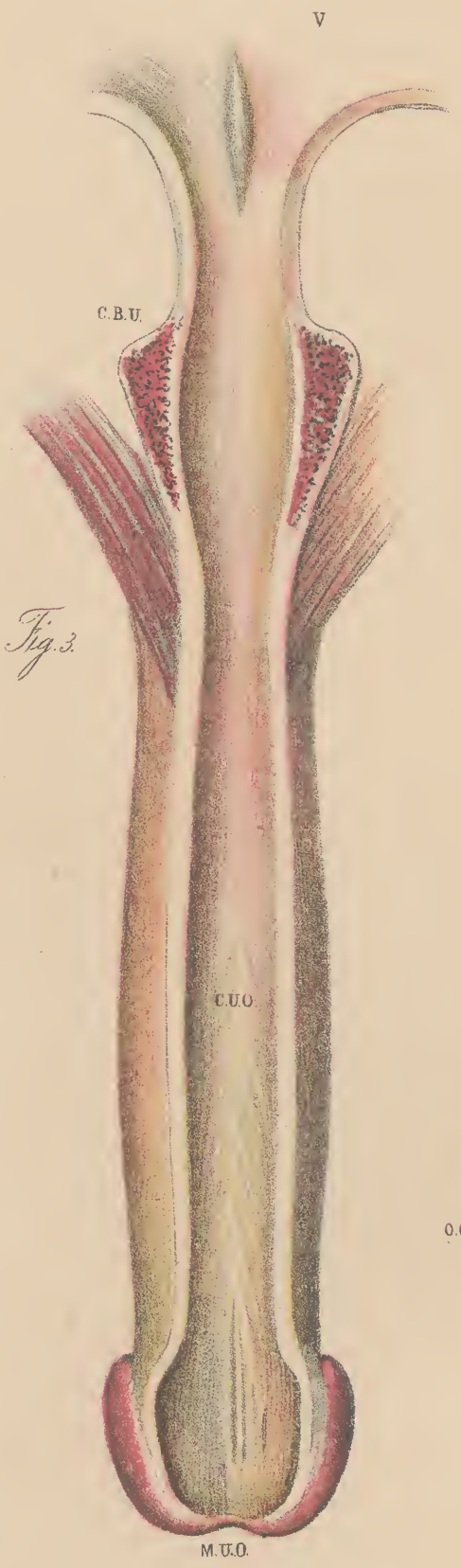


Fig. 3.

Fig. 3'

Fig. 3"

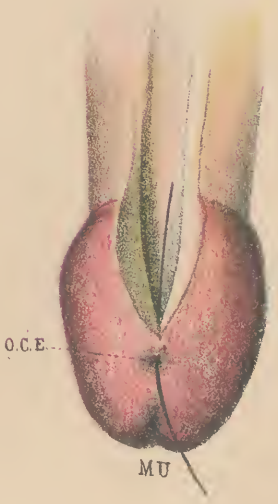
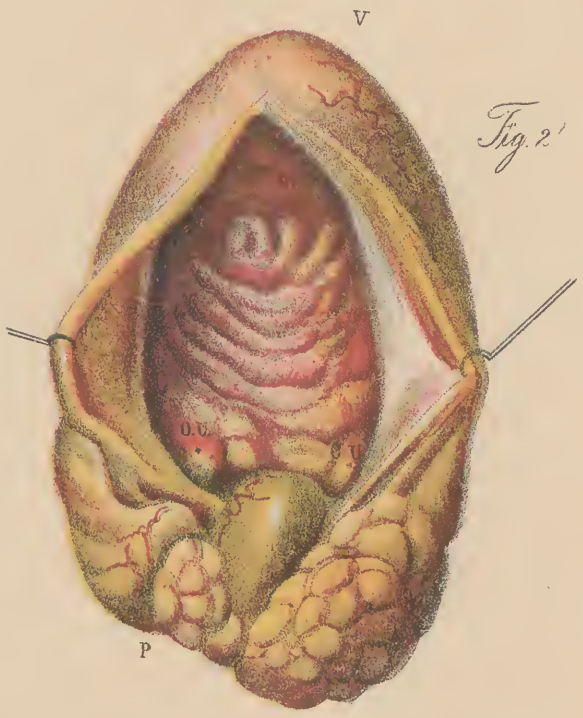
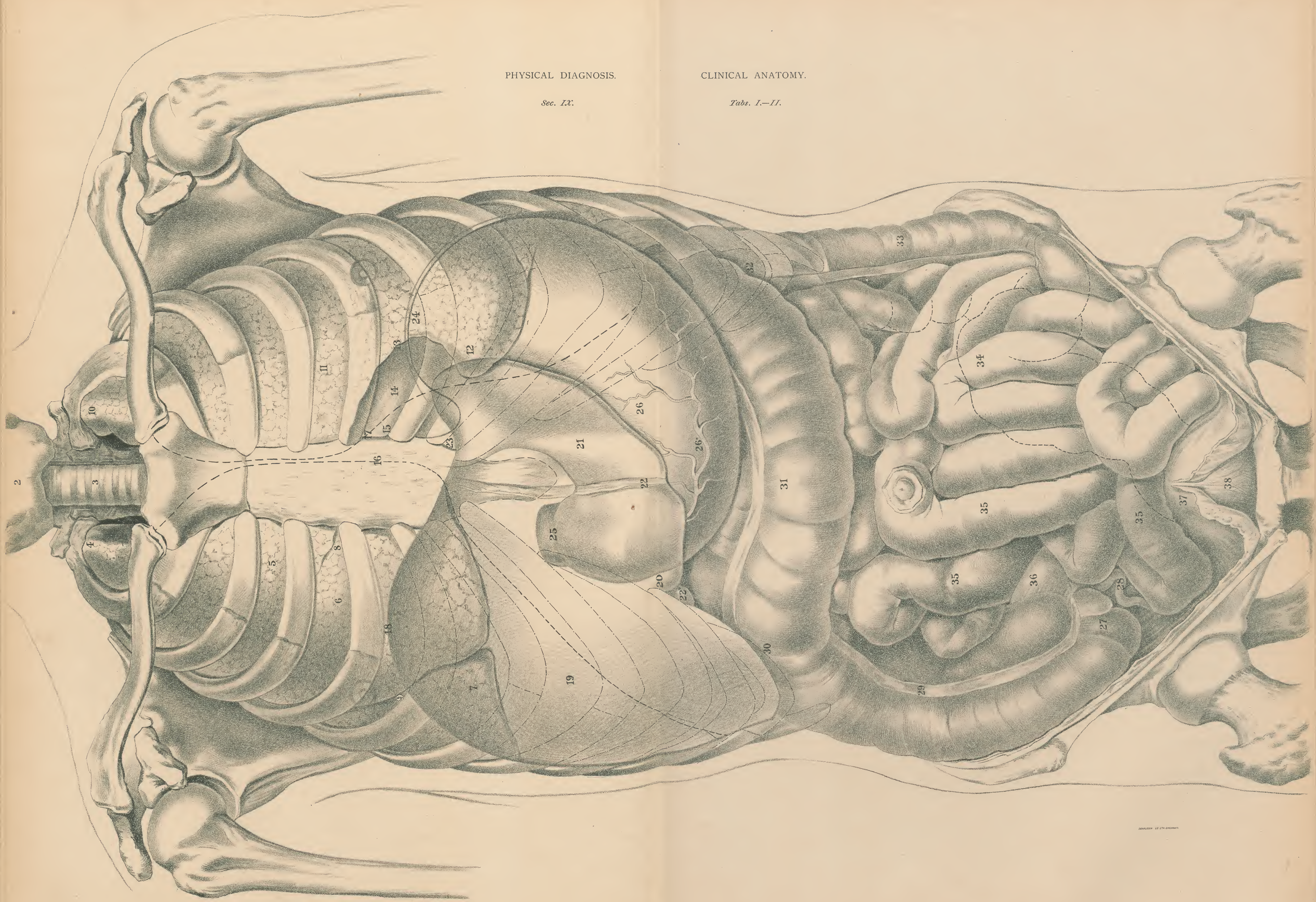


Fig. 2'



The Donaldson Lith. Co. Cincinnati.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PHYSICAL DIAGNOSIS.

CLINICAL ANATOMY.

ANTERIOR ASPECT OF THE ORGANS OF THE THORAX AND OF THE ABDOMEN, NATURAL SIZE, IN THEIR RELATION TO THE SKELETON AND TO THE TRUNK. (IN SITU.)

SECTION IX, TABLES I AND II.

THE lungs are represented in a state of expiration, and the hollow organs of the abdomen in a state of dilatation.

1. Larynx.
2. Thyroid gland.
3. Trachea.
4. Apex of right lung.
5. Superior
6. Middle } lobe of right lung.
7. Inferior
8. Superior } interlobular fissure of same.
9. Inferior
10. Apex of left lung.
11. Superior lobe of the left lung.
12. Lingual portion of the upper lobe of the left lung.
13. Cardiac fissure of the anterior border of the left lung.
14. Portion of the anterior side of the pericardium invested by the pleura.
15. Portion of the pericardium not invested by the pleura. (Proper place for pericardial paracentesis.)

Both localities indicate the place of absolute dullness of cardiac sound.

16. Anterior boundary of right pleura.
17. Anterior boundary of left pleura.
18. Superior, true boundary of the liver covered by the right lung.
19. Right hepatic lobe.
20. Lobus quadratus of liver.
21. Left lobe of liver.
22. Portion of suspensory ligament of liver.

*22. Fundus of gall-bladder.

23. Superior orifice of stomach.
24. Great cul de sac of the stomach, partly beneath the left lung.
25. Pyloric extremity of the stomach.
26. Part of the stomach impinging upon the epigastrium, and partly covered by the liver.

*26. Right gastro-epiploic artery, corresponding to the great curvature of the stomach.

27. Coecum.
28. Vermiform process.
29. Ascending colon.
30. Right flexure.
31. Transverse colon.
32. Left flexure.
33. Descending colon.
34. Sigmoid flexure covered by convolutions of small intestine.
35. Convolutions of small intestine ascending obliquely to the right.
36. Portion of the urinary bladder covered by the peritoneum.
37. Portion of bladder, not covered by peritoneum, projecting above the superior border of the pelvis, in a semi-distended condition.

PHYSICAL DIAGNOSIS.

The diagnostic means which physicians use in examining the sick are partly chemical, partly physical in their nature. The latter are those which are most frequently employed, and constitute the most important ones, at the present time.

When the physician places his finger upon the radial pulse in order to judge of its quality, or when he determines the bodily temperature by means of the thermometer; when by the ear or the stethoscope he endeavors to perceive the peculiarity of the sound or sounds produced by the organs of respiration, or circulation, the same as by tapping with the tips of his finger or the mallet, he elicits sounds from hollow organs and their contents, he performs certain acts, which are in their nature physical processes. These modes of investigation have been named, by common consent, *physical diagnosis*.

Not the physician who uses the most numerous and the most complicated instruments is the quickest and surest Diagnost, but he who uses his senses with the greatest skill and experience. To acquire these it is necessary to practice, methodically, the different modes of physical examination, and to train the senses to observe every phenomenon manifested in diseases, and to learn to *correctly* interpret their significations. The physical methods of examination, which require no particular instruments in their use, are those chiefly performed by the eye, INSPECTION, and by the hand, PALPATION. They, as a general rule, constitute the beginning of every examination methodically carried on.

All conditions of the body perceptibly deviating from those which experience has taught us to consider as normal, can very readily be proven to be alterations of the physical state normally existing in the body. They constitute the clinical signs of the diseased body.

It behooves the medical man to translate these signs in such a manner that they should become indications of anatomical lesions and altered physiological processes, in his hands, and thus raise clinical experience from empirical guessing to actual knowledge.

Inspection.

Very many diseases of the internal organs are connected with readily recognizable alterations of the external skin. They are sometimes so pronounced as to almost dispense with a further close examination of the disease. The importance of the inspection of the integument is therefore very evident. Still, care must be taken not to attach too much importance to it alone, unless the lesion is strictly cutaneous, and then when it involves only the integument.

Alterations in the condition of the skin are:

1. Changes of color.
2. Changes in the degree of moisture.
3. Œdema of the skin.
4. Emphysema of same.
5. Change of its temperature.

Modifications of the color of the skin are physiological and pathological.

Physiological are the normal peculiarities of skin in the different races and in the inhabitants of different zones; degree of exposure to the sun, rain, etc.

Pathological are the Cyanotic, the Icteric, the Bronze-color, the Gray, and the Red. Paleness of the skin invariably depends on the quantity and the quality of the blood circulating in the Cutis. It exists when there is either active contraction of the vessels or lack of blood; either through loss of blood (hæmorrhage), insufficient quantity of colored corpuscles, or insufficient quantity of hæmatine. Besides, in conditions of active contraction of the blood vessels (fainting, fear, etc.), by insufficient cardiac force, either temporary or permanent, from whatever cause; severe losses of blood by acute sickness or cachectic state; by suppuration or severe exudations, etc., pale color of the skin will be produced. The same may be caused by lack of reproduction of the blood, owing to diseases of the blood-producing organs, insufficient nourishment, etc. Certain substances capable of destroying the blood directly will, when introduced into the body, produce paleness of the skin.

Redness of the skin is due chiefly to causes contrary to those producing paleness.

Cyanosis or blue color of the skin. This color may pass through all shades, from purple to deep bluish black. The physiological cause is chiefly excess of carbonic acid in the blood, also an insufficient proportional quantity of oxygen. It indicates a venous kind of blood.

As the overfilling of the blood with carbonic acid is chiefly due, either to a grave disturbance of the functions of respiration or circulation, or to both at the same time, such a color of the skin will exist to a greater or less degree in extensive lesions of either

TABLES III AND IV.

1. Superior lobe of left lung.
2. Inferior lobe of same.
3. Interlobular fissure of left lung.
4. Superior lobe of right lung.
5. Inferior lobe of right lung.
6. Middle lobe of same.
7. Superior fissure of right lung.
8. Inferior fissure of right lung.
9. The stomach, which descends here rather more vertically than usual, is here represented in dark outline.
10. Spleen, in its usual distance from the vertebral column. It is here shown as following the direction of the ninth, tenth and eleventh ribs; and its relation to the lung, during expiration; also its relation to the left kidney.

[N. B. It must be remembered, that in reality the side presented here becomes visible only when the diaphragm has been removed.]

11. Stomach. The left kidney, its pelvic portion and origin of the ureter, is shown here as if not covered by the spleen. The right kidney is only indicated by dotted outlines.
12. Superior horizontal portion of the duodenum.
13. Descending portion of duodenum.
14. Inferior horizontal portion of duodenum.
15. Duodeno-jejunal flexure.
16. Liver.
17. Hepatic duct.
18. Cystic duct.
19. Ductus choledochus.
20. Head of the pancreas; body marked by dotted lines.
21. Ascending colon.
22. Descending colon.
23. Posterior portion of descending colon not invested by peritoneum.
24. Portion of the sigmoid flexure in the cavity of the left ilium.
25. Rectal portion of sigmoid flexure.
26. Course of rectum, in front of sacrum and coccyx, curving forward. The direction of its curves show that they finally pass, to some extent, into the regions of the sacro-spinal and sacro-tuberosal ligaments.
27. Terminal portion of the rectum descending below the apex of the coccyx.

CLINICAL ANATOMY.

In order to be able to determine the changes taking place in morbid conditions of the thoracic and abdominal cavities and their viscera, the normal localities of these organs and their mutual anatomical and physiological relations must be understood.

For inspection, percussion and auscultation, the boundary of each must be well defined and borne in the mind of the examiner; for only in this manner will he be able to distinguish between line and line, surface and surface, sound and sound. The following anatomical descriptions and the drawings in the plates are according to Luschka: (*Die Brust und die Bauchorgane.*)

ORGANS OF THE THORAX.

The lungs do not exactly correspond to the extent of the thoracic walls, for above they pass beyond their limits, whilst below they do not reach the extreme boundaries of the walls. Nor are they symmetrical. The right lung is shorter than the left, by so much space as the diaphragm curves upward on that side, whilst it is wider and more voluminous than this. The anterior border of the right lung slightly passes the median thoracic line and into the left cavity.

The right lung is divided by two fissures into three lobes, the left by one fissure into two lobes.

In the right lung, the longest fissure, which divides the upper from the lower lobe, runs at first in the posterior two-thirds of the intercostal space, between the sixth and seventh ribs, then passes down to the diaphragm.

The lesser fissure which divides the upper lobe in two segments, has its course bordering on the anterior half of the intercostal space between the fifth and sixth ribs. Thus the middle lobe of the right lung comes in contact with the diaphragm and assists in forming its inferior surfaces and borders as well as its anterior border.

The fissure of the left lung begins at a level with the posterior edge of the intercostal space, between the fourth and fifth ribs, and terminates behind the cartilage of the seventh rib. The upper lobe forms its upper border, but partakes very little in the formation of the lower border and the lower surface of the lung. This latter forms the lingual lobule.

The apex of the lungs partly projects above the upper

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the organs of respiration or circulation, or in the hindrance of their function by mechanical, chemical, or other impediments, either existing in the body already or introduced into it.

Icteric Color.

The characteristic color, called icteric, which may vary from a bright lemon to a brownish yellow, is very easily diagnosed. It may exist, like other abnormal colors, in some localities only, or diffused all over the body.

When biliary coloring matter enters into the circulation and is carried into the skin it stains the latter yellow, of different shades. The conjunctiva becomes most distinctly so, and even the lips will look yellow when its natural red color disappears upon pressure. The soft palate will likewise look yellow when the mouth is opened very wide.

A number of yellow coloring substances obtained from coal tar products produce a similar effect upon the skin.

Bronze-color.

Addison found that when the suprarenal capsules are destroyed, the skin turns a grayish yellow or bronze-color. Those parts of the integument which are usually exposed to the light are the first to be thus colored. Sometimes blackish or dark brown spots appear on some portions of the body at the same time. The conjunctiva is generally free from that color. Blue or gray color is produced by prolonged use of salts of silver internally, or by absorption (Argyria).

Copper salts very often produce a greenish tinge upon the face and other portions of the body.

A combination of colors is often met with in the skin of persons affected with diseases of the heart and hepatic complications. The color will be such as either the one or the other will preponderate in the combination: bluish-green, brown, even black, etc.

Change of form of a part of the body, or of the whole, can be ascertained by inspection. When surfaces which are known to be normally prominent, become less so, or become depressed, or when normal depressions become prominent and protrude, it is to be assumed that the change of surface is either due to increase or decrease of volume, with or without displacement, or only to displacement alone without change of volume of the part or parts.

Hollow organs are subject, physiologically, to change of volume, and with it displacement of surface; but that is only to a certain extent. Undue increase or decrease of volume in those organs, will indicate an abnormal condition. Parts of the body which normally maintain a definite volume and position will indicate an abnormal state, when there is a change in their volume or position.

Change of volume or position when injected.

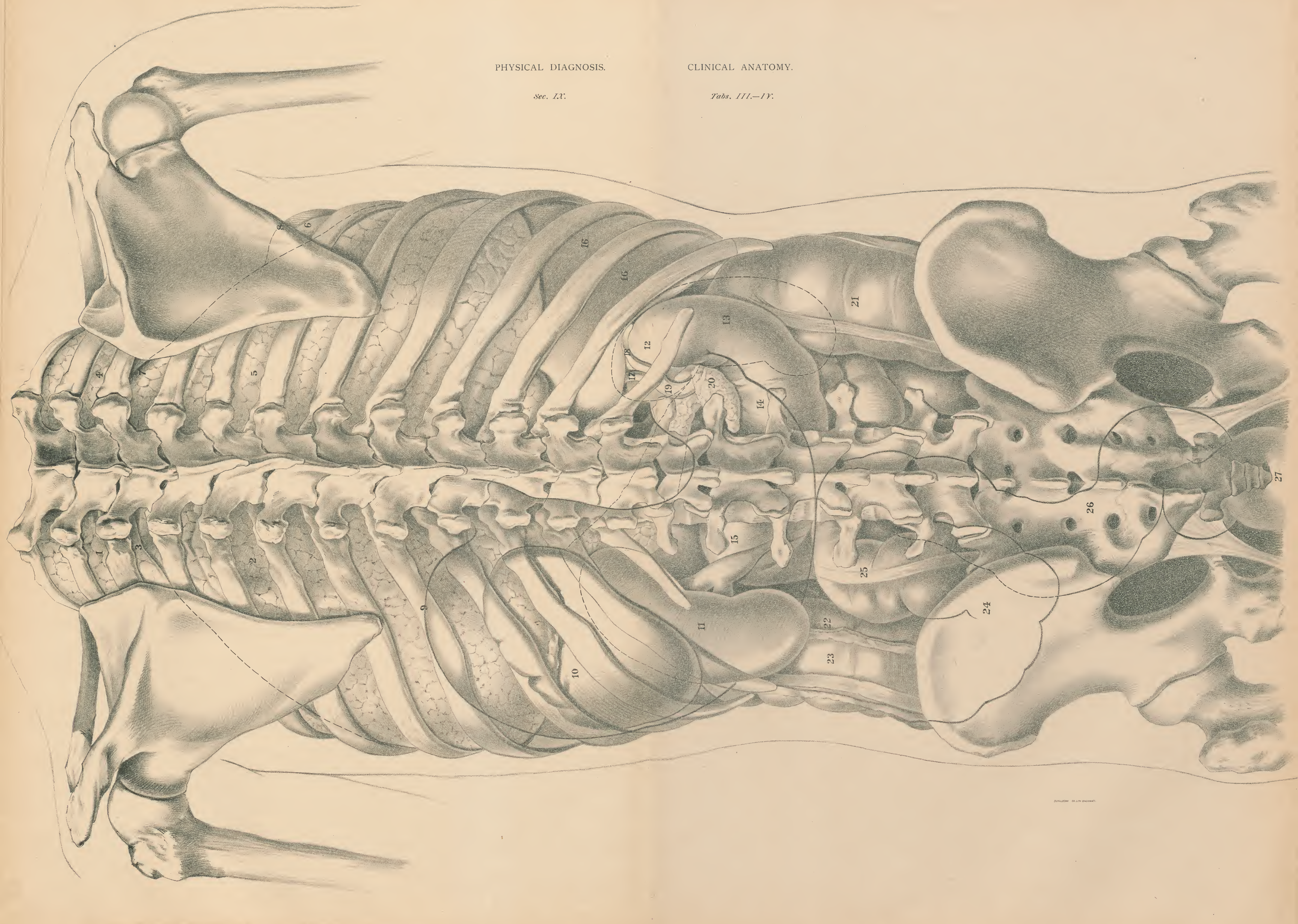
Slightly inflamed and infiltrated tissues, or infiltrated without being actively inflamed, hypertrophied, filled with solid or liquid neoplasms, with serum, pus or blood, will show an increased volume and a change of surface, by becoming more prominent than usual.

Inspection of the thorax.

The diseases of the thoracic organs exert such a powerful influence upon the form and movement of the chest, that by its inspection our attention is attracted toward that part which contains the diseased organ which we wish to examine.

When the thorax, which is normally wider at its base than at its apex, becomes flattened and uniformly narrowed; when the lower intercostal spaces, which are normally narrower than the upper, are as wide or wider than the upper, we may presume that a grave disease (like Tuberculosis, extensive hepatization or chronic Bronchitis) hinders the lung in its normal functions. The peculiarity of the shape of the thorax will awaken suspicion of the presence of pulmonic tuberculosis in its earliest stages.

When the thorax is depressed on one side more than on the other, drawn downward or extended longitudinally, the sub-clavicular fossa enlarged, whilst the ribs are drawn closer together, the spinal column more or less *scoliotic*, we may justly conclude that there has been an extensive pleuritic exudation; and the lung which was compressed was prevented from re-expansion,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orifice of the chest. The plane of elevation depends on the rise and fall of the first rib.

According to ordinary anatomical division it belongs to the lower lateral region of the neck; and is here, anteriorly and externally, covered and protected by the scaleni muscles. Ordinarily not recognizable by inspection, in severe vesicular emphysema it becomes perceptible, as a globular tumification, beneath the muscles and the skin. Percussion above the clavicle, in front, will not yield any satisfactory diagnostic results regarding conditions of the apex.

The external and internal surfaces of the lung terminate in the apex; the inferior surface constitutes its base. The internal being inaccessible to examination, it is here unnecessary to enter into farther detail.

The external or costal surface is the most extensive, and forms the convex surface of the lung. Posteriorly it occupies the lateral extent of the thoracic portion of the spine, and passes in front, partly, behind the sternum.

The costal portion of the right lung stretches behind the manubrium and behind the whole body of the sternum vertically down and beyond the median line. The left leaves the posterior surface of that bone at the sternal extremity of the fifth rib.

The vertical diameter of the external surface is shortest in front, and longest FROM A POINT ABOUT THE MIDDLE OF THE TWELFTH RIB TO THE UPPERMOST POINT OF THE APEX. The basal or phrenic surface is broadly semi-lunar downward and backward, and corresponding to the convexity of the upper surface of the diaphragm. It changes its planes with the movements of this structure. The lower surface is formed not only by the lower lobe, but also to the left by the lingual lower portion of the upper lobe; and to the right by a large three-sided portion of the middle lobe. Where the three surfaces meet, they form four borders: an anterior median, an inferior convex, an inferior concave, a posterior.

At the upper border of the manubrium sterni, the anterior median borders of the lungs are parted from each other to a distance equal to the width of the bone, and are situated immediately behind the sterno-clavicular articulation. From here they converge downward to the level of the second costal cartilage, where they almost meet, at the angle of Loudovic.

The right border passes the median line of the sternum to the left and extends either to the median border of the left two-thirds of the sternum, or, sometimes, even to the left sternal border.

The left border of the lung runs either along the left sternal border, or a little nearer to the median line.

From the second to the fourth costal cartilages both anterior borders run parallel to each other vertically downward, and separate only below that cartilage. From here the left border gradually passes into the inferior; the right passes almost vertically downward to the fifth cartilage, then turns downward and outward behind the sternum.

At the height of the fourth cartilage the left border of the lung turns almost horizontally outward, so that at the lower edge of the left costal cartilage it occupies its middle and external third; then describing a curve line—its concavity facing inward—it crosses the fourth and fifth intercostal spaces, passes between the inner and middle third of the left intercostal cartilages, where it sends the *lingual lobule* to partly cover the heart, and then passes into the left inferior border of the lung. This strongly diverging line of the left border forms an irregularly quadrangular space in front, which affords great facility for the percussion of the heart; for here a portion of this organ lies directly against the thoracic wall, without any interposition of air-containing tissue.

The concave notch of the lung is called the cardiac fissure or notch, and the triangular projection covering a portion of the heart, lingual lobule or lingual process.

THE PLEURA.

The pleural sacs essentially represent the form and size of the lungs; and are therefore asymmetrical. Each pleural sac consists of two layers or lamellæ; a visceral, covering the lungs and in immediate connection with the pulmonic parenchyma, and a parietal, or external, lining the internal thoracic wall. Both lamellæ stand to each other in relation of continuity, at the root of the lungs and pulmonic ligament, and everywhere their free, smooth and moist surfaces are in contact with each other.

The visceral pleura as an integral part of the lung has been included in the description of the pulmonic surfaces. Farther description of the pleura applies to the costal or parietal portion.

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even after absorption of the exudate, either by atrophy or attachment and pseudo-membranous formations.

Obliterated extensive pulmonic cavities also produce partial depression of the thoracic wall.

The existence of considerable deformities of the thorax, in consequence of curvature of the spinal column, indicate: an *emphysematous* condition of the lung below *the protruded portions* of the thorax, and a *compression* and sanguinous *plethora* below the *depressed portion*. Such a condition is associated with simultaneous dilatation of the blood-vessels.

When the sternal region of the chest is protruded and is barrel-shaped and enlarged in its antero-posterior diameter, whilst its vertical diameter is diminished, an emphysematous condition of *both* lungs will be found.

Emphysema of one lung will only protrude the affected side of the thorax, likewise a one-sided pneumo-thorax or one-sided pleuritic exudate. An emphysematous state of only a portion of the lung, which is mostly found in the anterior and upper border protrudes only that portion of the thorax.

Protrusion in the region of the heart indicates either an enlargement of the heart, chiefly the left, or accumulation of liquid in the pericardium.

Changes of surface of the thorax connected with the changed position of its organs of respiration.

Normally active respiration shows that the movement of the thorax, the innervation, and the contraction of thoracic muscles, and especially the distension of the lungs, conjointly constitute the physiological function of breathing. Any change in either of these processes carries it into the range of morbid phenomena.

Pathological types of respiratory movements.

1. Inspiratory dyspnœa is indicated by:

Long drawn inspiration, connected with great muscular effort, very often also with stretching of the spine and throwing back of the head, whilst the larynx forcibly descends.

The retarded entrance of the air during inspiration is indicated by a dragging, buzzing, sipping sound in the larynx. The expiration follows easy, short and noiseless.

It is generally produced by paralysis of the posterior crico-arytenoid muscles, by inflammation or œdema of the aryepiglottic folds, which close like valves by inspiration and part in expiration.

Pedunculated polypi of the rima glotidis, also certain spasmodic contractions, like epilepsy, hysteria, gallstone, colic, spasms of the glottis produce similar phenomena.

2. Expiratory Dyspnœa indicated by:

Long drawn, whooping and forcible expiration, produced by powerful contraction of the abdominal muscles, with flexion of the spine, followed by short, easy inspiration, takes place when there are polypi with long peduncles in the trachea, or croupous membranes, foreign bodies, in the larynx. In spasms of the diaphragm, and very often in morbus Brighti of a high grade, it also exists. In asthma, this dyspnœa is due to flatus of the lung (Biermer). In long standing emphysema, it exists also (F. Riegel).

3. Exaggerated *male* respiration indicated by:

Slight movement or insignificant protrusion of the upper part of the thorax, accompanied with great tension of the muscles. Exists in obliterated or much shrunk condition of the apex of the lungs. The lower portion of the thorax is that much more expanded, and the intercostal spaces and the diaphragm are that much more active. Tuberculous patients mostly present this type of respiration.

4. Exaggerated female respiration.

Found in the latter stages of pregnancy, and is expressed by strong inspiratory tension of the scaleni, sterno-cleido-mastoid and intercostal muscles, distension of the upper thoracic aperture and slight movement of the diaphragm. The lower ribs and the anterior abdominal wall are in an almost complete state of rest.

This type of respiration is found in paralysis, protrusion upward, or in very severe flattening of the diaphragm. In very extensive ascetis; when large tumors exist in the abdominal cavity or invade it from the pelvis. It is easily recognizable by simultaneous dis-

This constitutes two divisions, one of which stands in relation with the ribs and the lateral expanse of the thoracic portion of the spinal column, and with the diaphragm; the other passes like a partition-wall through this region of the thoracic cavity. Above, both are concerned in the formation of a blind sac, which projects at the inner border of the first rib above the wall of the thorax, and corresponds in shape to the rounded form of the apex of the lung.

As the upper extremity of the pleural sac rises above the first rib and is there connected with a number of structures, which are immovably attached, the apex of the lung is necessarily as immovable as the rest of the tissues there, and readily permits infiltration into its parenchyma to stagnate; and exudate externally, to form attachments to the surrounding tissues.

The lateral wall of the pleura is the most extensive division of that membrane. It lines not only the internal surfaces of the ribs and intercostal spaces, but also the lateral extent of the vertebræ; and extends in front behind the sternum. Its upper border is marked by the internal edge of the first rib. The lower border does not reach to the cartilagino-osseous limit of the thorax.

Those points where the pleura is reflected from the ribs upon the diaphragm must be regarded as its lower boundary. The line described by the reflexion of the membrane varies in height, and its direction is as follows:

On the left side it passes behind the external third of the bodies of the sixth and seventh ribs, but does not come in contact with the cartilages of the ribs, from the eighth to the twelfth.

On the right side it differs from the left only, that it descends behind the whole costal cartilage of the sixth rib, obliquely, and then continues in the same direction as on the left.

The internal walls of the pleural sac, the mediastinum, forms a sheath on each side, extending from the anterior wall to the posterior of the middle region of the thoracic cavity. It is perforated by the structures which enter and leave the lung at the root of the organ, and where the membrane, forming a double sac, is reflected upon the inner surface of the lung as its serous covering.

Above the root of the lungs the mediastinum connects the anterior and posterior pleura, and assists in forming the cul de sac of the apex of the lung.

Below the root of the lung it forms a continuity with the pleural portion of the diaphragm, which it also duplicates, and passes in a postero-anterior direction, at the lower extremity of the posterior border and the posterior edge of the lower border of the lung, to become the visceral portion of the membrane.

This duplicature is the so-called pulmonic ligament. It is triangular and attached by its apex to the root of the lung, and by the lower extremity of its posterior border, to the diaphragm. Portions of the mediastinum are attached to several thoracic organs by some connective tissues. With the lateral and partly anterior portion of the pericardium it comes in close contact; and assists in covering the trunk of the phrenic nerve.

Above the pericardium, to the right, it invests the external surface of the superior vena cava and vena innominata; to the left the external surface of the aorta.

The anterior mediastinal space is shorter than the posterior; it has the height of the sternum to the place of attachment of the xiphoid cartilage; and is unequal in width in its several portions.

The upper portion is triangular, its base facing upward, its apex downward, reaching the second rib, sometimes no lower than the first rib. In it are lodged the arch of the aorta, the origin of the large arterial trunks and the vena innominata. In the portion, from the second to the fourth rib, the two surfaces of the membranes lie very close to each other; but below that they diverge very much, chiefly on the left. The lower triangular space — its base facing downward and apex upward—contains a great deal of fatty cellular tissue, the heart and the pericardium, which is attached by that tissue to the anterior wall of the chest. The phrenic nerve and many lymphatic glands are also imbedded in it.

The posterior mediastinal space has the height of the thoracic portion of the spinal column. It is wider at the root of the lung than above or below it. Close to the spinal column and slightly to the left of the median line the descending aorta passes from the right of the third thoracic vertebra, downward, and then gradually moves to the median line. The œsophagus and the pneumo-gastric nerves descend to the right of the aorta, the former crosses the vessel and gradually passes to the left.

The thoracic duct ascends to the right of the aorta and lodges, higher up, between it and the œsophagus. To the right of this lies the vena azygos, into which empties the vena hemiazygos, after crossing the spine at a level with the eighth thoracic vertebra. The anterior portion of the upper division of the posterior mediastinal space is occupied by the lowest portion of the trachea and very numerous lymphatic glands. As the anterior and posterior mediastina meet at the roots of the lungs, the ingoing and outgoing structures of the organ are invested by both membranes.

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tension of the lower part of the thorax, with a high position the heart occupies in that cavity.

5. Unilateral type:

Decrease of respiratory movement of one side and enhancement of the other side. Effusion in one pleural cavity, loss of elasticity in one lung, shrinking of one, pressure upon one side of the thorax from without (tumors, etc.) cause an increase of respiratory labor and greater expansion of the opposite side, provided it be in a healthy condition.

6. Incomplete inspiration; type of new-born babes:

All conditions under which the free ingress of air into the respiratory passages and the lung-tissue is prevented, produce enormous efforts of respiratory movements without effect.

The clavicle, the sternum, and the upper ribs are raised, the larynx descends, the glottis is widened, yet the respiratory organs are not filled. The pressure of the air upon the internal surface of the thorax is diminished. The external pressure drives the yielding portion of the thorax in.

The clavicular fossa is depressed, the same is the case with the intercostal spaces, the xiphoid cartilage and the adjacent costal cartilages. When the bronchi are obstructed or contracted, respiration is more frequent; when the larynx or glottis is obstructed or contracted, respiration is very slow. The inspiration and expiration are accompanied by a snoring or resonant rhonchus, according as the *atresia* is in the pharynx or in the larynx. The diaphragm is driven upward by every inspiration, the anterior abdominal walls sink in and the upper border of the liver rises high up.

Such a condition, when in existence for some time, causes a narrowing of the thorax. Such a respiratory type is to be found in broncho-stenosis, tracheo-broncho-stenosis and unilateral atelectasis; also in all contractions of the pharynx, larynx and trachea; in severe catarrh of the respiratory passages, and in the asthmatic attacks of emphysematous patients.

CIRCULATORY PHENOMENA OBSERVABLE BY INSPECTION.

Pulsation.

All movements of the heart produced by its systole, or coincident with this, are designated as pulsations.

The most important is the apex beat, a movement executed by the apex of the heart. With this is connected the so-called visible motion of the heart; pulsation perceptible between the umbilicus, xiphoid cartilage and the arches of the ribs in front, between the second and fourth costal cartilages; the pulse of the large arterial trunks, carotids and subclavian, also of the jugular and venous pulsations sometimes perceptible in the neck.

The apex-beat is normally found in *the fifth intercostal space*, between the papillar and parasternal lines, occupying a space of about two and a half inches in diameter; noticeable during systole as a slight temporary protrusion of the thoracic wall.

It may vary, to a great extent, even in healthy persons, both in position and degree, according to posture and respiratory condition of the individual, as well as with the peculiarity of structure of the chest and position of its viscera.

Pathologically, the apex-beat may become enormously modified both in regard to locality and strength. It may shift its position from the second to the ninth left intercostal spaces, and from

Sec. IX. *Tab. V.* **BOUNDARIES OF VISCERA.**

Fig. 1.

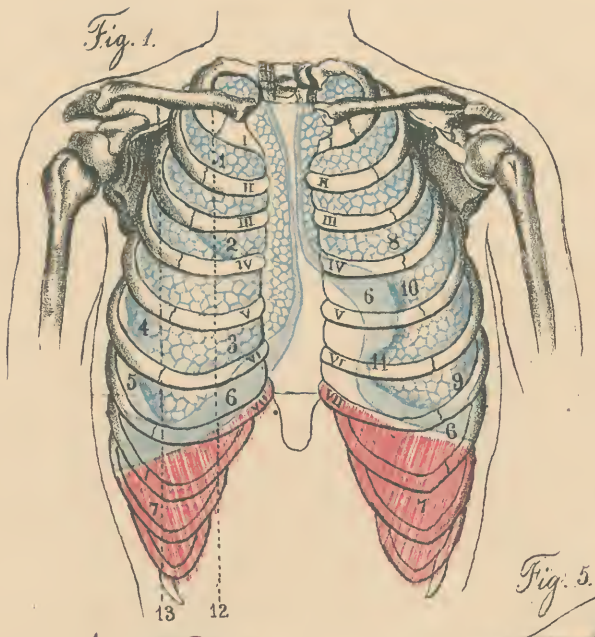


Fig. 2.

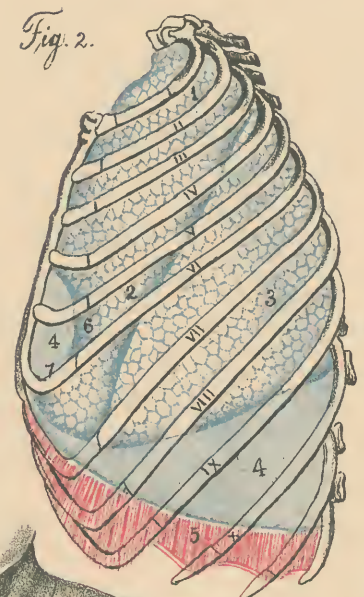


Fig. 3

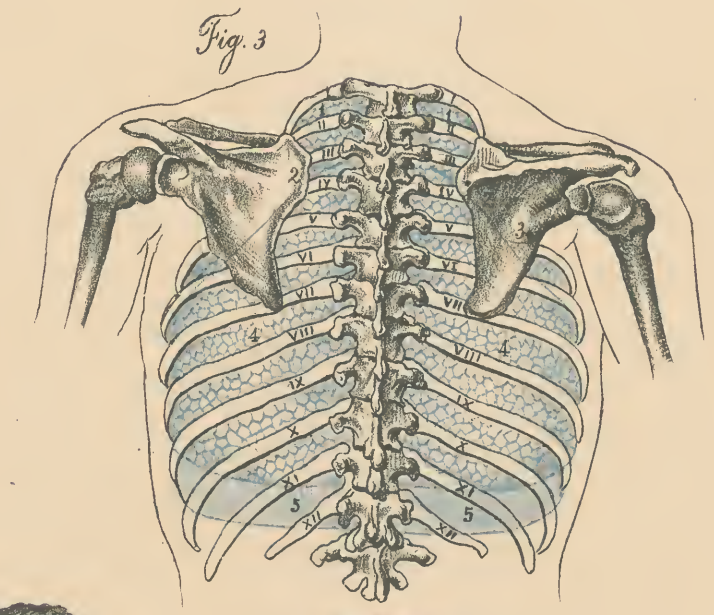


Fig. 4

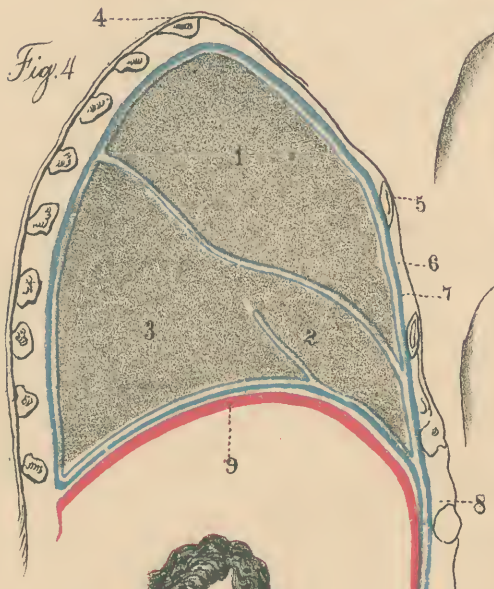


Fig. 5.

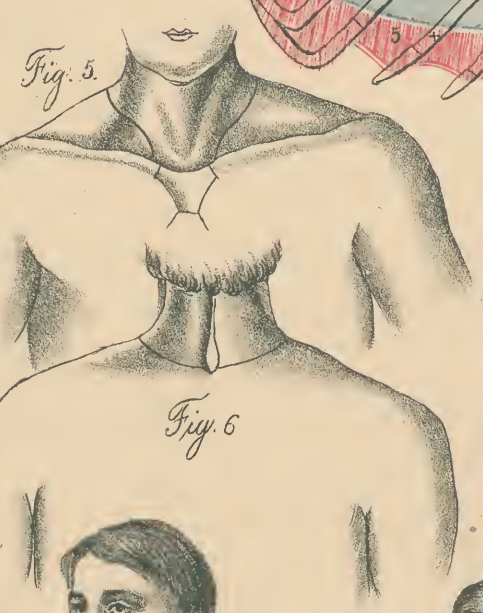


Fig. 6

Fig. 7.

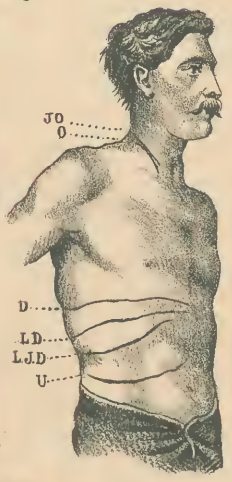


Fig. 8

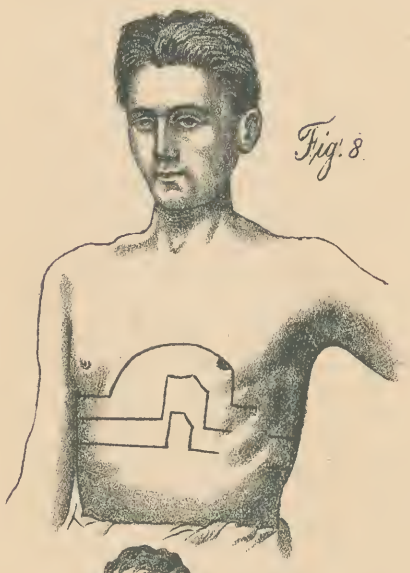
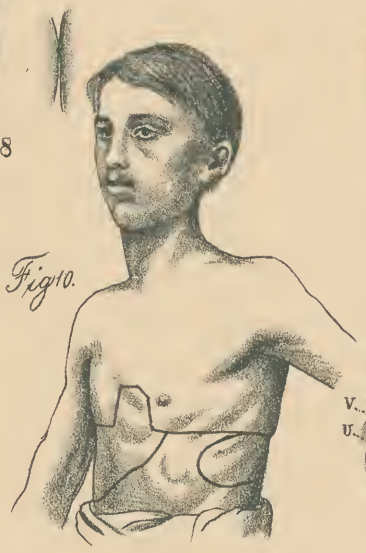


Fig. 9

Fig. 10.



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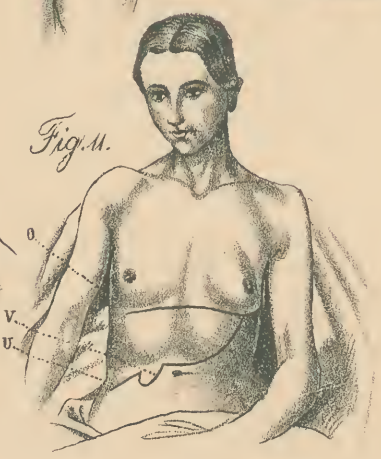

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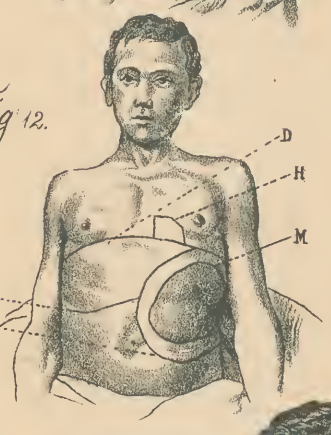

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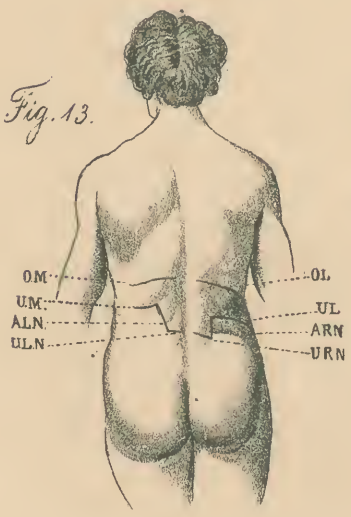
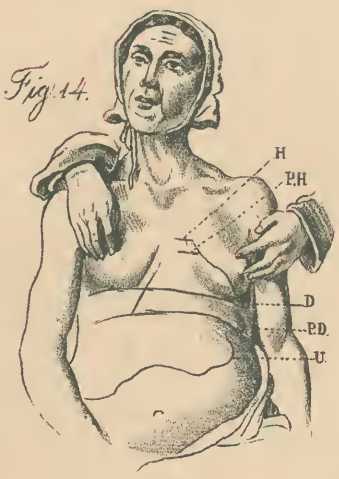


Fig. 14.



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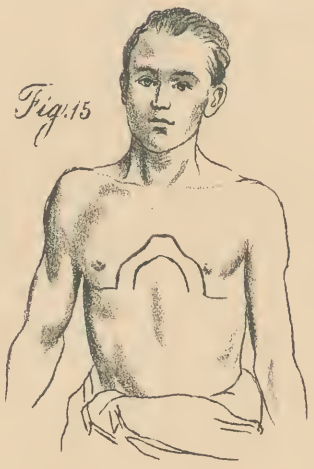

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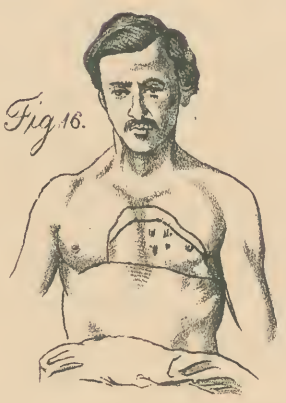

Fig. 17.

Fig. 18

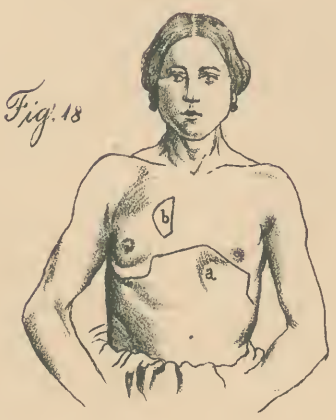

Fig. 19

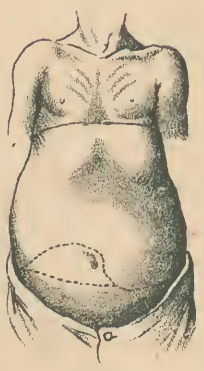

Fig. 20

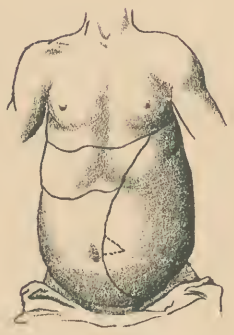

Fig. 21.

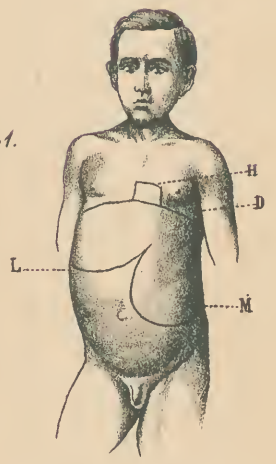

TABLE V.

ANTERIOR THORACIC ASPECT.

FIG. 1.—*The boundaries of the different viscera are marked in different line markings. The organs are differently colored.* (1), upper lobe of right lung; (2), right interlobar fissure; (3), middle lobe of right lung; (4), inferior interlobar sulcus; (5), lower lobe of right lung; (6), pleural and complemental space; (7), diaphragm not covered by pleura; (8), upper lobe of left lung; (10), cardiac notch; (11), lingual lobule; (12), parasternal line; (13), papillary or mammillary vertical line.

FIG. 2.—*Left lateral aspect.* (1), upper lobe; (2), interlobar fissure; (3), inferior lobe; (4), pleural space; (5), diaphragm; (6), cardiac notch; (7), lingual lobule.

FIG. 3.—*Posterior thoracic aspect.* (1), upper lobe; (2), interlobar fissure; (3), division in the sulcus; (4), lower lobule; (5), pleural cavity.

FIG. 4.—*Vertical section of the thoracic cavity.* (Right half.) (1), upper lobe; (2), middle lobe; (3), inferior lobe; (4), posterior extremity of second rib; (5), anterior extremity of second rib; (6), pleura pulmonalis; (7), costal pleura; (8), complemental space; (9), diaphragm; (10), portion of same not covered by pleura.

FIG. 5.—*Boundary for percussion of the apices of the lungs* (*Gerhardt*).

FIG. 6.—Same behind.

FIG. 7.—The same, shifting in changed positions of the body. (O), upper boundary of the lungs; (I O), upper boundary in inspiration; (D), position of diaphragm; (L D), diaphragm whilst the person lies on the left side and breathes quietly; (L I D), diaphragm in left recumbent position during inspiration; (U), lower boundary of lung.

FIG. 8.—Position of the diaphragm and boundary of area of dull cardiac sound, in quiet but deep inspiration and expiration.

FIG. 9.—Area of cardiac dull sound, in erect posture, in right and left recumbent position in a youth fifteen years old. (a b c), cardiac dull space; (M L K), empty cardiac sound; (e h g f), recumbent position on the right; (a n i), recumbent position on the left.

FIG. 10.—Arch formed by the area of dull sound of the heart, liver, and spleen.

FIG. 11.—Dull sound over the gall bladder in catarrhal icterus, where the tense organ projects beyond the anterior border of the liver.

FIG. 12.—*Splenic tumor in intermittent fevers.* (D), diaphragm; (H), area of cardiac dull sound; (U), inferior boundary of the liver; (M), area of splenic dull sound; (M R), border of spleen during a fever paroxysm.

FIG. 13.—Area of dull sound of the kidneys. (O M), upper splenic region; (O L), upper boundary of liver; (U M), lower splenic boundary; (U L), lower hepatic boundary; (A L N), external boundary of left kidney; (A R N), external boundary of right kidney; (U L N), inferior boundary (left); (U R N), inferior boundary of right kidney.

FIG. 14.—*Change of position of diaphragm and increased area of dull sound in insufficiency of the tricuspid valve, before and after puncture for ascites.* (H), region of cardiac dull sound; (D), position of diaphragm; (U), inferior border of liver before puncture; (P D), position of diaphragm after puncture.

FIG. 15.—Area of cardiac dull sound in hepatization of the right lower lobe of the lung. The same after resolution.

FIG. 16.—Cardiac area in pericarditis in erect and recumbent postures.

FIG. 17.—Shrinking of the left lung, dislocation of the heart, and high position of the diaphragm.

FIG. 18.—Cardiac hypertrophy and aneurism of the ascending aorta. (a), cardiac dull sound; (b), dull sound of the dilated aorta.

FIG. 19.—Area of dull sound in enlarged liver from echinococcus hepatis. Within the space between the dotted lines the liver was lying beneath the intestine.

FIG. 20.—Splenic tumor of a patient affected with leukæmia.

FIG. 21.—Hepatic cirrhosis in a fifteen-year-old youth from drinking alcoholic liquors. (H), cardiac dull sound; (D), diaphragm; (L), lower boundary of liver; (M), spleen.

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from the 3d to the 7th right intercostal space, in front. In hypertrophy and dilatation of the left ventricle, without stenosis of the aortic opening, the ventricular systole may cause a strong protrusion of the thorax in the 6th or 7th intercostal spaces. It will be noticeable, according to the position of the diaphragm, in the space between the sternum and the nipple, either to the right or to the left. In this lesion there is, usually, besides the protrusion, a strong vibratory movement of the thorax to the left. In extreme cases of the kind the whole lower portion of the thoracic wall is thrown to the left. When the enlarged heart is situated horizontally there is usually depression in the 3d or 4th intercostal spaces near the sternum. In hypertrophy of the right ventricle, with dilation, the lower part of the sternum and the adjoining left costal cartilages are pressed forward during ventricular systole. When both ventricles are hypertrophic and dilated the intercostal spaces corresponding to the apex may be raised, the lower portion of the sternum protruded, and the whole lower part of the thorax pushed to the left.

In dilatation and hypertrophy of the right ventricle, there is in the 2d, 3d, or 4th intercostal spaces either protrusion or reversely falling in of the thoracic wall. In weak heart extensive pericardial effusion causes the apex-beat to disappear. When the heart is strong it remains visible.

Percussion and Auscultation of the Thorax.—As most of the objective symptoms of the thoracic organs have been described in the text, in their respective places, only the mechanical manipulations in the process of diagnosis will be here described.

Percussion is carried on either immediately, that is, by tapping with the tips of the fingers directly upon the surface of the body under examination, or intermediately, that is, by striking upon any resonant substance placed between the patient's body and the finger of the examiner. In former years plates of ivory of divers shapes were employed for that purpose, and were called *plessimeters*. It has been found that tapping upon one or two fingers placed upon the thorax answered the same purpose as the plessimeter, and was withal handier and more to the point. An obsolete method of percussion is tapping with a small mallet upon the plessimeter. Although any of the methods may be properly employed, the simplest is certainly the most rational, and that is finger-percussion. By percussion the differences of resonant or non-resonant parts of the body, also the amount of resistance to the percussing instruments, are to be ascertained. Viscera and parts of viscera situated beneath covering walls of cavities give rise to sound phenomena in conjunction with those of the walls themselves when these are made to sound by percussion. The following sound phenomena have been, empirically, found to exist and classified:

1. All fleshy parts which contain no air yield a dull or weak, muffled sound, similar to that evolved by tapping with the finger upon the fleshy parts of the arm or thigh.

2. Fleshy organs containing no air or any liquid, and enclosed in other fleshy parts, yield a dull sound.

3. Every sound evolved by percussion upon the thorax or the abdomen differing from the one described, is due to the presence of liquid or air-like substance either in those cavities or within the viscera in them.

Theoretically, compound sounds and single percussion sounds must be differentiated. Compound sounds consist of both the vibrations of the thoracic walls and of the viscera, and differ:

1. According to the greater or lesser state of elasticity of the walls.

2. According to the quantity or state of the air or liquid in the viscera.

The pulmonary parenchyma gives rise (according to the degree of tension) to more or less regular vibrations of the air, which may become a tone or be only a sound.

Accordingly the air may here be considered as the source of sound, which may be full or loud, dull or muffled. Since normally the thoracic sounds are not produced by regular vibrations, they do not assume the dignity of *tones*, but are only sounds, which have been, conventionally, divided into clear and dull sounds, with their intermediate varieties. Dullness of sound is graded according to the thickness of the substance, which contains no air, situated immediately beneath the thoracic wall.

Tympanitic and Non-tympanitic Sounds.—*Tympanitic* or *drum-like* are called those sounds which come near being *tones*, and are due to greater regularity of vibration than mere sounds. The less regular the vibrations the farther are the sounds from the tympanitic type. For instance: when a portion of an intestine, which is neither distended nor blown up, is percussed, it will give a tympanitic sound; but when it is distended or strongly blown up by air or gases, it will yield a non-tympanitic sound on percussion.

Causes of the Sounds.—The air confined within a definite space in an organ will be put in vibration by percussion; the vibrations being the more regular the less they are interfered with by the wall of the cavity which contains the air, or by other substances contained within the same cavity. A tightly drawn wall of, or a solid substance in a cavity containing air, interferes with its vibrations, and produces irregular waves which yield a non-tympanitic sound. A flabby wall or soft substances interfere less with the regularity of the vibrations, the sound will come near being that of a musical sound or a *tone*, and will have a tympanitic cast. This physical property of air confined in spaces of a definite dimension, and the thickness of the walls of such spaces, will exactly determine the character of sounds producible by percussion. A thin wall with little air in the cavity will yield quite a different sound from that of a thick wall and much air in the cavity; for it is evident that a thin wall will interfere sooner with vibrations of air, by its being easier stretched, than a thick wall, etc.

The pitch of sounds depends on the sound wave put in motion. It is so much higher the nearer it comes to being a tone or of a tympanitic type.

In closed cavities the height or depth of sound depends on the length of the sound wave; in open cavities it depends on the length of the wave and on the width of the orifice of the cavity. For instance: when percussion is made upon the relaxed cheek, the lips being closed, the height or depth of the sound produced depends on the size of the cavity of the mouth. In percussion upon the cheek, with open mouth, the height of the sound will be greater the more the mouth-cavity is diminished or the less wide the orifice is made (*Niemeyer*).

Physiological Percussion.—The aim of percussion upon the surfaces of the thorax and abdomen is to ascertain the position of the several viscera situated within those cavities by the alternate clear and dull sounds which are evolved thereby. The respective viscera emit sounds proper to their peculiar structure and their relative position to the walls of those cavities. These are the typical sounds. But as one organ or a part of an organ overlaps another, or a part of another, the cardinal sounds become modified, and intermediary sounds are thus produced. This makes it necessary that the force of the percussion should be modified according to circumstances. Forcible percussion is necessary when an organ situated very deep beneath the wall of the cavity, or when the wall is very thick, is to be examined. Gentle percussion is indicated when a comparatively thin air-containing medium is situated in front of a solid part of an organ. In order to localize the several sound-boundaries in the thorax a number of conventional lines have been established. Thus the *sternal line* runs from the superior semi-lunar notch of the sternum to the apex of the xiphoid cartilage. The *parasternal* line begins at the junction of inner and middle thirds of the clavicle, and runs

TABLE VI.

Sphygmographic Tracings (F. RIEGEL).

FIGS. 1, 2, 3, normal adult male's pulse. The vertical line (a b c) is the directing line. It is not always perpendicular, but is inclined sometimes to the right and sometimes to the left. Each sphygmographic curve is made up of the ascending line or limb (g c), the descending (c h), and the point or pivot (c), which latter constitutes the summit or apex of the angle. When the ascending line contains secondary elevations it is called *anacrot*. When the descending limb has elevations it is called catacrot. (*Landois.*) According to the number of elevations in the descending line it may be termed dicrotic, tricrotic, tetracrotic, etc. In good health the ascending line rises vertically, without any interruption, for the artery is swiftly dilated with every ventricular systole, the lever of the instrument rises in a straight line, and speedily turns down to form the descending line. This marks the general character of the pulse, for it is made up of a number of curves succeeding each other and forming one or more waves. The healthy pulse describes three curves or elevations in the descent of the lever, with great regularity, and is called a polycrotic pulse. It indicates the tension of the artery and the density of the blood (see text). The descending line has two elevations (c d), the first designating the first secondary rise of the blood pressure, the second the following secondary rise, such as elasticity of the vessel, etc. In Figs. 1, 2 there are two elevations showing the normal dicrotism. These are always perceptible if there be any force in the blood-current at all (d). Figs. 4, 5, 6, typical pulses of convalescents from typhoid fever. Both secondary elevations are plainly visible. In delirium the second elevation is very prominent, whilst in old age it is the reverse. It becomes a slow pulse. Figs. 7, 8, 9, 10, 11, 12, typical pulses of old age as found by MAREY. The ascending line is not always straight, and the angle which the descending line forms at the summit is often a right or an obtuse one. Fig. 7 is especially characteristic of the pulse of old age. Fig. 9, a type of paralysis of the arterial wall. 10, 11, 12 are all anacrotic. Figs. 13, 14, 15, 16, 17 show the difference between the curves of pulse during inspiration and those formed during expiration. 13, 14, which are from the same patient, when respiration was quiet but deep, whilst 15, 16, 17, pulses of anæmic convalescents, show modification during quiet and superficial respiration. The changes in form consist in that the inspiratory pulses are all smaller, show deeper and higher secondary elevations, and far more insignificant elevations from elasticity, etc., than those in the act of expiration. Fig. 18 shows the inequality of two respiratory acts. Figs. 19, 20 are what *Kussmaul* calls *pulsus paradoxus*, found in verrucous mediastinal pericarditis. The pseudo membrane stretched from the sternum to the pericardium compresses and draws the aorta during ventricular systole and thus produces irregularities of circulation in that vessel. The inequalities in respiration produced by changes in the upper respiratory passages are shown in 18, 19. Figs. 20, 21, 22, 23, 24, 25, fever pulses. 20 indicates reduction of tension in the arteries taking place in fever (*Marey*). 21 is a normal pulse to show the contrast between it and the fever curve, which shows the gradual loss of dicrotism in the pulse. Fig. 26, convalescence from typhoid fever, with a temperature 38.4 centigrade. Fig. 27, acute rheumatism, with a temperature of 40.3 centigrade. Fig. 28, a pulse corresponding to a temperature of 39.3 centigrade. Fig. 29, pulse with a temperature 38.6 centigrade. Fig. 30, pulse with temperature 39.3 centigrade. Figs. 31, 32 show pulses corresponding to temperatures of 39.3, 40.0 centigrade. Figs. 33, 34, 35, pulses of old people in typhoid fever. Figs. 36, 37, 38, 39, 40, 41, 42, 43, pulses observed in typhoid-fever patients several days in succession. Each figure indicates the temperature during the observation. Figs. 44, 45, 46, 47, convalescents, showing increase of dicrotism in the pulse as the temperature decreases. Figs. 48, 49, pulses of consumptive patients, the temperature varying from 37.0 to 39.0 centigrade, and frequency of 96. Figs. 50, 51, pulse of a consumptive with remission of the fever. Figs. 52, 53, of the same patient during the fever. The first corresponds to a temperature 38.6, the second to a temperature 37.8 centigrade. Figs. 54, 55, a very consumptive patient. There was great variation of temperature in a few hours. Figs. 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66 show pulses with great variations of temperature and respiration in a number of infectious and inflammatory fevers. Fig. 67, pulse produced by the action of amyl nitrite. The tension is here seen to be enormously reduced whilst the frequency of the pulse is correspondingly increased. Fig. 68, with a temperature of 37.1 centigrade whilst the frequency is only 40 per minute. In Fig. 69 the frequency is 160, temperature 37.5 centigrade The contrast is great between the two graphics. In one there is great arterial tension and very slow pulse; in the other very low tension and very frequent pulse. Figs. 70, 71 show a very rigid wall and great resistance to the cardiac systole in lead colic. The secondary elevations are low but very close to each other, frequency 71 per minute. Fig. 72, vascular tension in Bright's disease, with compensatory hypertrophy of the left ventricle. Figs. 73, 74, 75, 76, cases of acute nephritis, in all of which there is high arterial tension. Digitalis produces high arterial tension and causes a slow pulse, but at the same time increases the force of the heart. Sudden disappearance of pleural exudate has a similar effect. Fig. 78, a case of mitral insufficiency. Fig. 79, mitral insufficiency with incomplete compensation, and beginning degeneracy of the muscular tissue of the heart. The pulse is here very irregular. Figs. 80, 81, 82, 83, cases of stenosis of the mitral valve. It produces a contrary effect upon the pulse than in mitral insufficiency, it becomes very small, and the secondary elevation is correspondingly very insignificant. Fig. 84, the pulse of a young man of nineteen years, affected with insufficiency of the aortic semi-lunar valves. Fig. 85, that of a twenty-three-year-old man, with the same affection, but with derangement of compensation and other complications. Fig. 86, a similar disease, but with derangement of the compensatory progress, and complicated with mitral insufficiency. Fig. 87, a case of aortic insufficiency. Fig. 88, *aortic stenosis*. The peculiarity of the pulse is the exceedingly steep ascending lines with great amplitudes. This is produced by gradual formation of hypertrophy and dilatation of the left ventricle. Fig. 88 shows a contrast with a pulse in mitral stenosis. Fig. 89, a case of simple hypertrophy of the left ventricle without aortic insufficiency, in a case of Bright's disease. The ventricle, in this disease, is increased in volume, and has the force to dilate the artery and the semilunar valves also to repel the blood, and cause a full secondary elevation. Figs. 90, 91, 92, 93, 94 show irregular pulses in diseases of arteries and in cardiac arhythmia. Fig. 95, a pulse in cerebral meningitis. In this disease there is enormous irregularity of the rhythm, and inequality of the motor forces of circulation.

vertically downward to an indefinite extent. The *costo-clavicular* line, left, beginning at the inner extremity of the clavicle, runs down in front to the 11th costal cartilage. The line to the right corresponds with its fellow on the left. The *mammillary* lines run from the point of junction of the external with the middle third of the clavicle, through the nipple downward, on each side.

As in the living body the thorax and abdominal viscera shift their positions in the respiratory act, the diaphragm becomes the pivotal point, and by this the normal from abnormal movements of the viscera are to be gauged. By percussion of the thoracic and abdominal viscera is to be ascertained, when the diaphragm becomes immovable, whether the cause of it is in the diaphragm itself or in the organs above or below it.

In order to obtain as much space as possible in percussion of the chest the following rules are usually adopted:

1. To expose the clavicular region, both arms and shoulders are drawn backward, the chest thrown forward, as near an erect position of the chest as possible taken, and the hands or forearms crossed on the back.

2. To expose the scapular region, raise both arms above the head, bend them at the elbows, cross forearms and place the right hand upon the outer aspect of the left arm, and the left hand upon that of the right arm.

3. To expose the suprascapular region, cross forearms, with bent elbows, in front of the chest, grasp the left side with the right hand, and the right side with the left hand.

4. To expose the subscapular region, cross the arms with bent elbows in front, the right hand grasping the tip of the left shoulder, the left hand the tip of the right.

As the normal position of the thoracic organs are described in Sec. IX, Tables 1, 2, 3, 4, it is only necessary to describe now the respective sound-territories of those organs in their normal position and in quiet breathing.

1. *The Area of Cardiac Dull Sound* extends from the upper borders of the third ribs in front down to the upper borders of the seventh ribs to the left, forming a triangle, with its base upward and obliquely to the right, and its apex downward and to the left of the sternal edge.

2. By far the greatest part of the heart in front is covered by the anterior and lower borders of the lungs, yet the dull cardiac sound is normally not changed by that circumstance. Percussion on each side of the cardiac boundaries yields a clear, non-tympanitic sound. Outside those portions of the lungs which are bounded by the sound-territories of the liver and the stomach, both sides have the same sound everywhere. Any change in its character can readily be noticed.

3. The apex of each lung, whether by direct percussion upon the clavicle or indirect upon the clavicular fossæ, yields the same sound, unless the pectoral muscles are enormously hypertrophic, or any tumors or other enlargements of the tissue are lodged there, and muffle the clear sound. The changes perceptible in pathological conditions of the lungs, on percussion, have been described in the text in their respective places.

Auscultation of the Thorax.—Auscultation is carried on either by placing the ear directly upon the portion of the body to be examined or by means of one kind or another of acoustic apparatus (stethoscope, etc.), (see Sec. IX, Table VII).

All sounds perceived by percussion are produced by the same mechanism, that is, by causing the parts to vibrate by the stroke of the finger or instrument. Sounds obtained by auscultation are produced in a two-fold manner:

1. By movements of liquids or gases in tubes and other cavities.

2. By friction produced by movable surfaces touching each other when in motion. The first series of sounds produced in the cavities of circulation and respiration are the most important in the technique of auscultation, both by their great variety and their indicating the conditions of the most important functions of the body.

The circumstances of sound-production by moving liquids and gases in cavities are as follows:

1. Membranes placed in the line of movement of liquids or gases are put in motion, *vibrate*. The cardiac valves and the vocal cords are examples of that kind.

2. By narrowing tubular cavities or by contraction of their walls, whereby the even current of the liquid or gas is changed into an intermittent or rhythmic motion, sound is produced.

3. Disturbance produced in a current meeting with a stationary obstacle produces sounds.

4. Contraction or vibration of a tubular wall when it forms a curve.

A. The movement of liquids or gases in tubes or tubular cavities is without sound as long as their diameter is uniform, or when it is alternately wide and narrow in succession. In the latter case it is indifferent whether the walls are even or uneven.

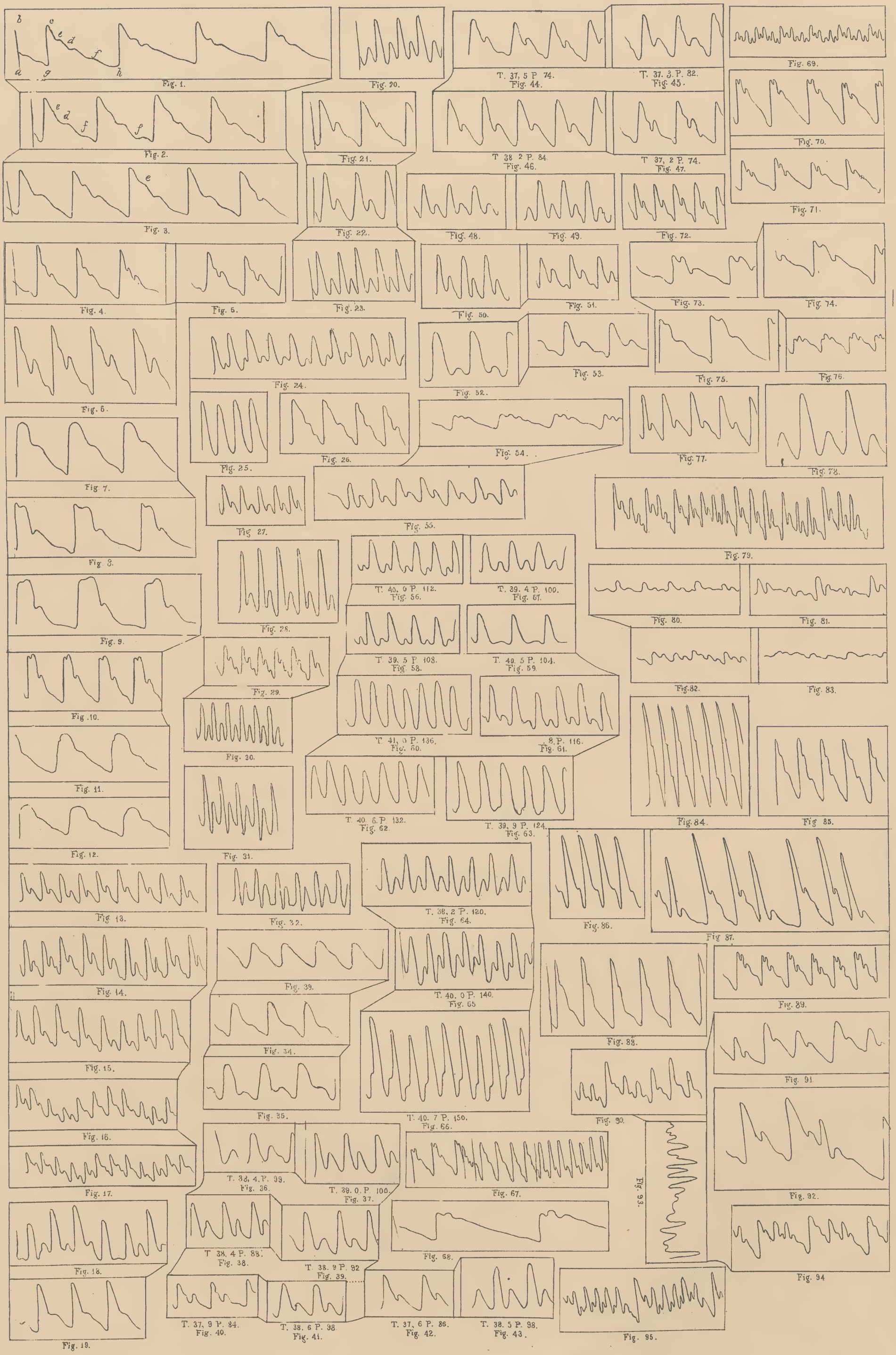
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Fig. 35.
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Fig. 36.
T. 39. 0. P. 100.
Fig. 37.
T 38. 4 P. 88.
Fig. 38.
T. 38. 9 P. 92
Fig. 39.
T. 37, 9 P. 84.
Fig. 40.
T. 38. 6 P. 98.
Fig. 41.
T. 37, 6 P. 86.
Fig. 42.
T. 38. 5 P. 98.
Fig. 43.
T. 37, 5 P. 74.
Fig. 44.
T. 37. 3. P. 82.
Fig. 45.
T 38 2 P. 84.
Fig. 46.
T 37, 2 P. 74.
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Fig. 55.
T. 40. 0 P. 112.
Fig. 56.
T. 39. 4 P. 100.
Fig. 57.
T. 39. 5 P. 108.
Fig. 58.
T. 40. 5 P. 104.
Fig. 59.
T. 41, 0 P. 136.
Fig. 60.
8 P. 116.
Fig. 61.
T. 40. 6. P. 132.
Fig. 62.
T. 39. 9 P. 124.
Fig. 63.
T. 38, 2 P. 120.
Fig. 64.
T. 40. 0 P. 140.
Fig. 65
T. 40. 7 P. 150.
Fig. 66.
T. 39. 0. P. 100.
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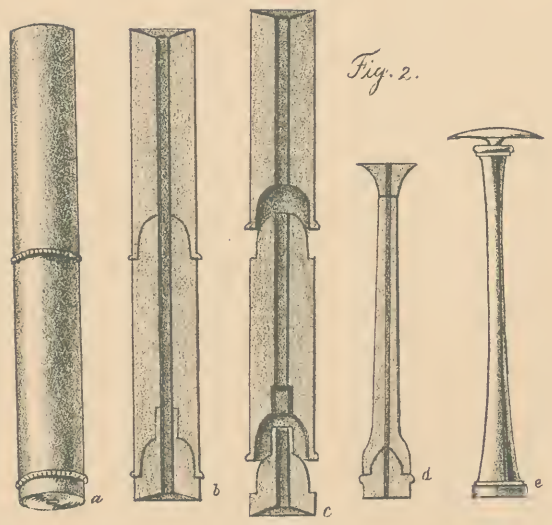

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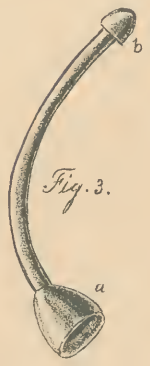

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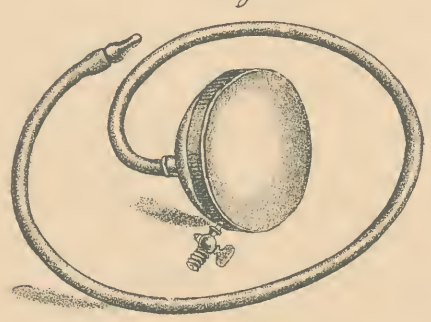

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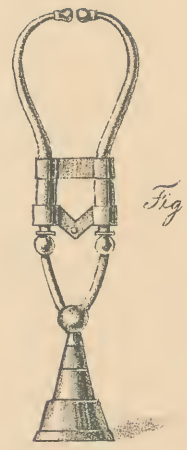

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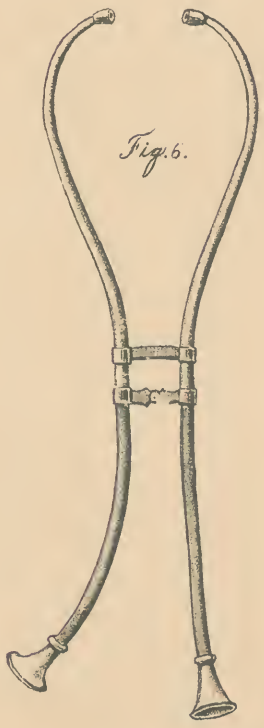

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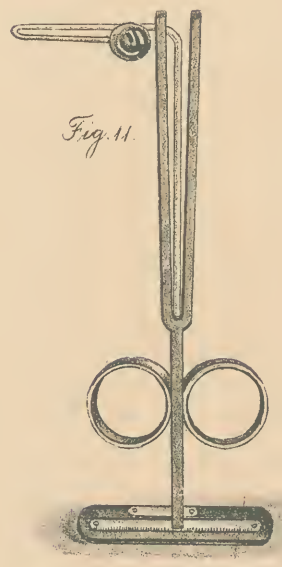

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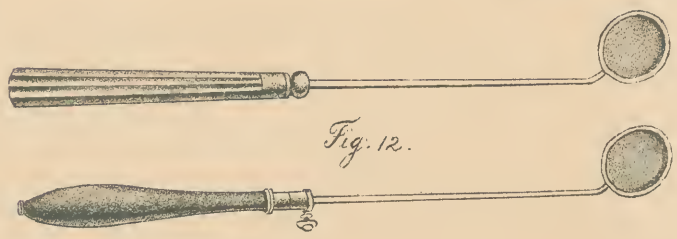

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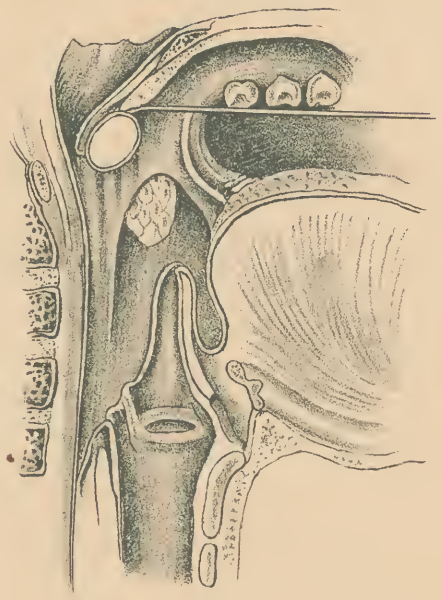

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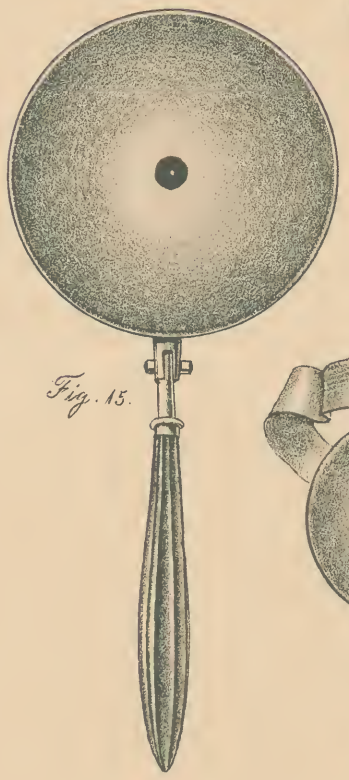

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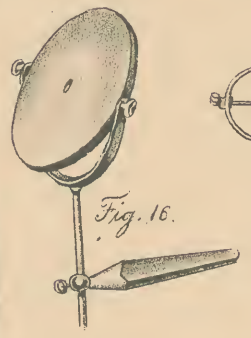

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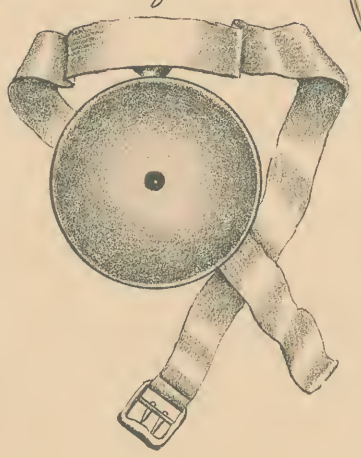

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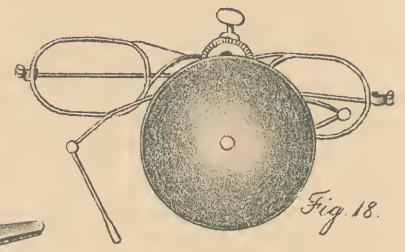

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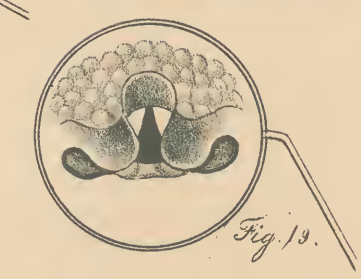

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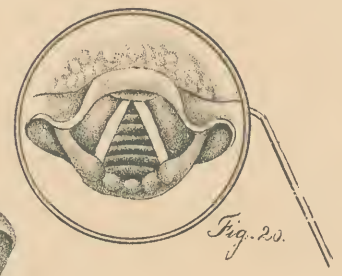

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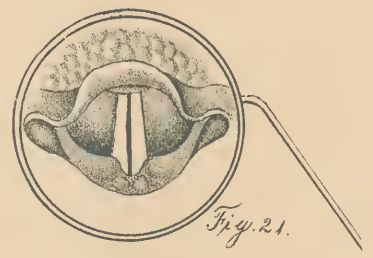

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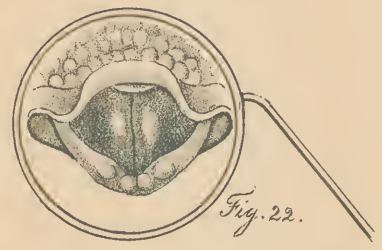

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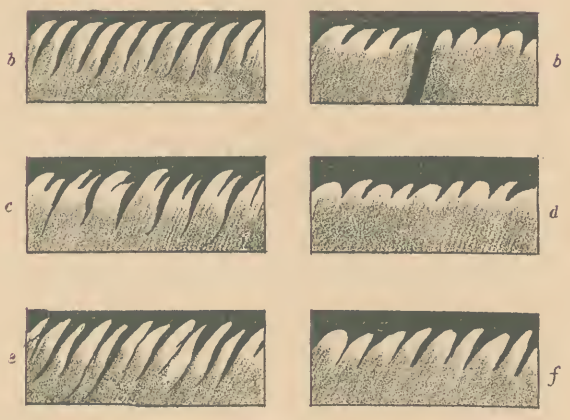

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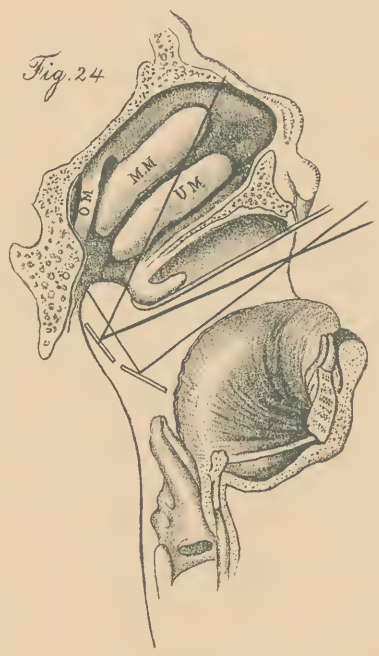

Fig. 25.

Fig. 26.

Fig. 27.

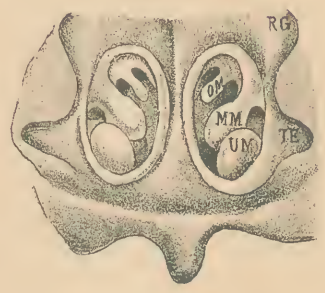

TABLE VII.

PHYSICAL DIAGNOSIS.

FIG. 1.—Paul Niemeyer's stethoscope. A solid cylinder made of very dry pine wood. Fig. 2.—Several forms of stethoscope. a b c, Laenec's; a, external surface; b, internal cavity; c, the several parts of the apparatus (one-fourth of its natural size); d, common stethoscope, with obturator (one-fourth of its natural size). Stethoscope, with convex ear-plate (one-third of its natural size). Fig. 3.—Voltolini's instrument, consisting of a funnel of hard pine wood, adjusted with a rubber tube about thirty or forty centimeters long; at the extremity the ear-piece or a hard rubber cap, mushroom shaped, is screwed on. *Gruber* and *Voltolini* claim great acoustic properties for this stethoscope. *Hueter* states that when a fine soft rubber membrane is tightly drawn over the orifice of the funnel and placed upon a large artery, a humming sound is heard, which is synchronous with the ventricular systole. It constitutes an *angioscope*, which is of especial use in insufficiency of the aortic semi-lunar valves. Some smaller arteries may also be auscultated with the instrument. Fig. 4.—Stethoscope of Koenig. It consists of a plano-convex capsule. The plane surface (the one to be placed upon the patient's chest) is covered with two soft rubber membranes, between which air is blown in to form a cavity with bi-convex walls. The acoustic rubber tube enters into the center of the convex surface of the capsule. The same maker proposes to adjust a number of tubes to this capsule so that several persons may auscultate at the same time. Fig. 5.—A stethoscope to be used by both ears at the same time. It is much in use in the United States. It has little or no advantage over the common wooden stethoscope in general use. The chief objection to it is that all sorts of sounds are heard and thus destroy the general effect of the sounds to be diagnosed. Figs. 6, 7.—Allison's differential stethoscope, offers no particular advantage over the ordinary instrument. Figs. 7, 8.—Somatoscope of Hueter and Waldenburg. They are ingenious playthings more than anything else. Figs. 9, 10.—Tuning-fork and resonance-block of Baas, to serve as a phonometer in percussion. Fig. 11.—The phonometer of Baas. Fig. 12.—Laryngoscopic mirrors, with a solid handle (a), with an adjustable handle (b). Fig. 13.—a b c d e f, several forms of mirrors; g, a mirror with a micrometer scale. Fig. 14.—Position of the laryngoscope in the pharynx; the uvula is gently raised and pushed backward, the mirror occupying an oblique position, whilst the handle of the instrument is horizontally held against the crowns of the back upper teeth. Fig. 15.—Convex perforated reflector (one-half of its natural size), movable backward on a joint screw. Fig. 16.—Szermak's reflector, with a tongue depressor adapted to the handle. Fig. 17.—Kramer's bandage, with reflector for the forehead of the operator. Fig. 18.—Smeleder's eye-glasses and adjusted reflector. Fig. 19.—View of the epiglottis, as seen with the laryngoscope. Fig. 20.—View of larynx during inspiration. Fig. 21.—Same in phonation. Fig. 22.—Same during strong expiration. Fig. 23.—a b c d e f, flame waves, produced by the human voice (according to *Klemm, phonoscopia*). Fig. 24.—Rhinoscopy. Fig. 25.—Pharyngoscopic mirror of Voltolini. Fig. 26.—Palate hook of Szermak. Fig. 27.—Rhinoscopic view. (o m), (m m), (u m), superior, middle, and lower nasal conchæ; (R G), Rosenmuller's fossæ; (T E), Eustachian tube.

B. Sounds in tubes are produced only when they become narrower in a definite locality, for thereby the even current is changed into an intermittent, rhythmical.

C. The sounds of the heart and arteries, also those of expiration, are produced by pressural current. Those of the veins and in inspiration by a suction current.

D. *Nature of Sounds, and their Causes.*—Many sounds heard in auscultation are simply oscillatory. These become amplified or complex by the presence of a membrane, in a narrow portion of the tube or cavity, and which takes part in the formation of sound.

Such membranes may have the form of a tongue (such as the vocal cords), and will impart to the sound a whistling character, or it may be in the shape of simple floating membrane (such as the movable vascular valves). Both are caused to vibrate by the moving liquid or gas.

Fremitus is called the tangible vibration of its particles produced by a sounding body, simultaneous with sound. Such is the fremitus that is produced by organ pipes, etc. When the vibration is very intense it will extend to substances in connection or contact with it.

Qualities of Sounds in Auscultation.—1. Indefinite sounds, such as produced by active contraction of the walls of a cavity. Such are: the sounds of ventricular contraction, that of vessels, and some of the respiratory organs.

2. Sounds of the stroke of the gas or liquid upon the walls when it turns an angle.

3. Incomplete vibrations of the walls, such as that of valves.

4. Murmurs (*bruits*). They may be simple oscillatory murmurs, such as the cardiac, arterial, placentar, inspiratory, laryngeal, and vesicular murmurs.

5. Whistling murmurs (amplified oscillatory), such as stenotic murmurs of the aorta, or that produced in expiration.

6. *Oscillatory-vibratory Sounds.*—Such as the rasping, clinking murmur in aneurism; or rough, buzzing, rattling.

7. Complex sounds, as in aneurismal sacs, or rales.

8. Tones, such as produced by rhythmic vibrations of membranes, the cardiac valves, the vocal cords.

9. Amphoric sounds, highly complex, such as cliqueti metallic, or rattling of coins; amphoric resonances.

Simple sounds are seldom associated with fremitus; oscillato-vibratory, or whistling sounds, are occasionally associated with it. Tones and all rhythmic vibrations are nearly always accompanied by fremitus.

Auscultation of the Cardiac Region.—A powerful apex beat sometimes becomes distinctly audible, (*a*) by cliqueti metallic, metallic ring, (*b*) in the region of the stomach; in pneumo-thorax in cavities of the lungs, etc., by an amphoric sound.

Cardiac Sounds, the name given to a great variety of sounds without much discernment between sounds and tones. They are produced by the valves, the heart, and arterial walls. Physiological analysis has demonstrated that only the second cardiac sound is of valvular origin. The first sound is complex, of valvular and muscular derivation. From a diagnostic point of view the valvular part is the most important. It differs so far from the second sound, which is purely valvular, as it is not a sudden but a gradual stroke. The auriculo-ventricular valves are not simple flaps that are closed by the pressure of the column of blood but highly complex structures, standing in connection with both the auricles and ventricles, a *fact* which allows them to regulate the entering and leaving column of blood. The sound production consists of two acts, the *initial* and the *terminal* valvular tension.

Nature of Cardiac Sounds.—With normal sounds are usually connected those which are, strictly speaking, extra cardiac, such as are produced by the systolic action of the large arteries (the pulmonary and the aorta). The locality where sounds are produced are: the basis of the heart with a rhythm of ⌣ —, and the apex with a rhythm of — ⌣. The first tone is always the same, the second tone a different one. The first tone is produced by the contraction of the pulmonary artery and the aorta, and by the systole of both ventricles and the vibration of the auriculo-ventricular valves. The second tone is produced by the closure of the semi-lunar valves. The topography of these tones are:

1. The auriculo-ventricular valve-sounds are audible in the fourth intercostal space, about one and one-half to two inches to the left of the sternal edge of that side.

2. Sound of the aortic ostium, audible over and about the sternal articulation of the third rib.

3. Of the descending aorta, on a line between the sternal extremity of the third rib and the sternal extremity of the second right intercostal space; on a line from the apex of the heart to the right clavicle.

4. The pulmonary artery: the sternal extremity of the second right intercostal space, about a half an inch from the left sternal edge.

In other localities the sounds are only conducted by liquids in the thorax, etc., and are not so distinct.

The character of the changes which may befall, under certain circumstances, these sounds are: division of sound and change of intensity. Division of sound consists in duplication of the tone. Instead of one, two or more are heard, produced first by a synchronous closure of valves existing in very deep inspiration; second, by the valvular edges not closing at the same time, by unequal contraction of the muscles of a valve or unequal tension of its flaps. The intensity in the second sound, depending on the force with which the column of blood closes the valve, is modified by greater or lesser weakness of such current, or by the obstacles it meets in its forward movement.

Murmurs or indefinite sounds exist in great quantities in functional disturbances and lesions of the heart.

Changes of Sounds in Cardiac Lesions.—These consist of permanent lesions of the valvular apparatus, which lead to disturbance of the valvular work and abnormal distribution of the blood. Derangement of valvular play will produce inequality in the character of sound produced by the valves; thus, when one valve emits a tone the other only produces a murmur or a blowing sound.

Disturbed or insufficient closure of a valve, of course, will leave an open space for the blood to flow backward, to regurgitate. Contraction of an orifice and change into a rigid, narrow opening, without any valvular closure, of course, will produce unequal distribution of the blood in the circulation. As a rule, this leads to compensatory hypertrophy of the organ in the locality of the lesion. Pathologically considered, stenosis and insufficiency are usually associated, and hence auscultation of the diseased heart will offer symptoms bespeaking of one or the other especially. Complications in heart diseases are so common that a number of secondary sound phenomena may mislead the inexperienced auscultator. Primary cardiac lesions affect almost exclusively the left heart, very seldom the right. In the right heart the tricuspid valve is usually the only part diseased, and only in secondary form; most frequently from lesions in the left half. Affections of the valves of the pulmonary artery but very seldom exist, except in the fœtus (*P. Niemeyer*). Practically, the diseases of the mitral valves, the tricuspid, and the aortic semi-lunar are the ones to be most studied, for they constitute the most frequent cases for clinical observations.

Lesions of the Mitral Valves.—Insufficiency.—The blood regurgitates into the auricle when that valve is unable to perfectly close. Stasis, at first in the lesser circulation, later in the whole body, takes place. Compensatory hypertrophy of the left usually ensues, and re-establishes quite a good arterial pulse. Sound symptoms during the stage of regurgitation are: systolic murmur associated with fremitus. This murmur extends from the apex to the axilla. The second tone, that of the pulmonary artery, is increased in strength. The apex beat is stronger but far diffuse.

Mitral Stenosis.—The narrowing of the auriculo-ventricular opening prevents complete filling of the ventricular cavity. At the same time there is regurgitation of the blood from the ventricle into the auricle as in insufficiency; the consequence of this condition is dilatation of left auricle, which prevents complete emptying of that cavity and atrophy of the ventricle, which also becomes disabled, and can not throw sufficient blood into the aorta; the semi-lunar valves also become atrophied. The arteries will, therefore, indicate these conditions. In insufficiency the pulse is little changed. In stenosis the pulse is permanently very small. There will be arhythmic action in stenosis with insufficiency: the systolic murmur will be long drawn and slightly snoring, over the apex—strong fremitus. Very strong second sound of the pulmonary artery, weak second

TABLE VIII.

TOPOGRAPHIC ANATOMY. PERCUSSION.

FIG. 1.—*Area of Dull Sound of the Heart.* (1), lesser space; (2), greater space; (3), area of cardiac resistance; (4), boundary of lesser area of liver sound; (5), boundary of lower border of left lung. Fig. 2.—Position of the heart. (1), heart; (2), right lung; (3), left lung; (4), complemental pleural space. In this is shown that a number of organs situated beneath and below the lung modify its normal, clear, non-tympanitic sound. Fig. 3.—Anacrotic venous pulse of the internal jugular (*Frerrichs*). Fig. 4.—Relative position of the aorta to the œsophagus (posterior view). (1), œsophagus; (2), thoracic aorta; (3), lungs; (4), trachea; (5), thoracic duct; (6), vena azygos; (7), left subclavian artery; (8), innominate artery; (9), diaphragm; (10), abdominal cavity. Fig. 5.—Position of the stomach (posterior view). (1), stomach; (2), spleen; (3), spleen; (4), kidney. Fig. 6.—Stomach (anterior aspect). (1), stomach; (2), liver; (3), heart; (4), lungs; (5), complemental space; (6), transverse colon. Fig. 7.—*Boundary of percussion of the stomach.* (1), greater; (2), lesser area of dull sound of the liver; (3), greater; (4), lesser area of dull sound of the heart; (5), resistant point; (6), lower border of left lung; (7), pulmono-hepatic sound; (8), boundary of stomach. Fig. 8.—*Anterior position of the liver.* (1), liver; (2), gall bladder; (3), heart; (4), right lung; (5), left lung; (6), complemental space; (7), pleural cavity. Fig. 9.—Posterior position of the liver. (1), liver; (2), complemental space; (3), kidney; (4), spleen. Fig. 10.—Area of dull sound of the liver (in front). (1), greater; (2), lesser area of the dull liver sound; (3), greater; (4), lesser area of the heart sound; (5), resistance of the heart; (6), lower border of the left lung; (7), angle of pulmono-hepatic sound. Fig. 11.—Area of dull sound of the liver (behind). (1), greater; (2), lesser area of dull sound of the liver; (3), kidney sound; (4), hepatico-renal dull sound (posterior). Fig. 12.—*Posterior position of the spleen.* (1), spleen; (2), complemental space; (3), left kidney; (4), right kidney. Fig. 13.—*Area of splenic dull sound.* (1), splenic area; (2), renal area of dull sound; (3), lateral border of left lung; (4), greater; (5), lesser area of cardiac sound; (6), left lower boundary of dull sound of the liver covered by that of the lung; (8), spleno-pulmonic sound; (9), spleno-renal dull sound. Fig. 14.—Posterior position of the kidneys. (1), left; (2), right kidney; (3), spleen; (4), liver; (5), descending colon; (6), ascending colon; (7), complemental space; (8), reno-hepatic angle; (9), spleno-renal angle. Fig. 15.—Posterior boundary of kidneys. (1), dull sound-area of the right kidney; (2), lesser area of dull sound of the liver; (3), larger hepatico-renal angle; (5), area of dull kidney sound; (6), area of splenic dull sound, spleno-renal angle.

tone of the aorta. Apex-beat diffuse, area of dullness very wide, pulse very small.

Lesions of the Tricuspid Valves.—Usually produced by stasis from lesions of the mitral valves. Regurgitation from the right ventricle into the auricle diminishes the stasis in the smaller circulation, yet the unequal distribution of the blood in the venous system will be visible in the pulsation of the jugulars. The same sound will be audible as in similar lesions of the mitral valves; and added to it will be systolic murmur between the five costal cartilages on the right. The second pulmonary tone will be intermittently strong.

Lesions of Aortic Valves—Insufficiency.—The blood wave regurgitating into the ventricular cavity produces reverberating oscillations, continuing in the aorta and readily perceptible in the carotid and even in the radial artery. Hypertrophy nearly always increases the volume of the left ventricle; the powerful ventricular systole causes, by forcing a large volume of blood into the aorta, distention and gradual rigidity of the arterial wall; there will be: diastolic humming murmur with a slight tone over the left ventricle, and extending to the jugular, chirping and systolic fremitus in the arteries. First cardiac sound muffled. Apex-beat deep, more to left, diffuse. Area of cardiac dullness long. Arterial diastolic murmur strong.

Stenosis of the Aortic Opening.—By disturbing the systolic blood wave moderate ventricular dilatation and hypertrophy are produced, also weakness and slowness of the pulse. The narrowing of the orifice may be so great as to give rise to a whistling sound or tone. If only murmur exists it is associated with fremitus. There are: Systolic blowing over the basis of the heart, extending over a large area; systolic fremitus over the apex; weak 2d sound of aorta; apex beat slightly increased and little diffuse; area of dullness somewhat increased in every direction; thin, dragging pulse.

A number of transitory changes of sound exist in transitory disturbances of the heart and its valvular apparatus. The following rules for auscultation of sounds and murmurs are given by *Paul Niemeyer* (*Percussion and Auscultation*), as obtained by experience.

1. *Over the apex* (left ventricle). In systole: A tone indicates integrity of the mitral valve. A murmur indicates relative insufficiency; *with a strong second tone*, absolute insufficiency of the mitral valve.

In diastole: A tone indicates normal state of aortic valves; a murmur, when it begins during the systole, indicates stenosis and insufficiency of the mitral valve. When only during diastole, indicates insufficiency of the aortic valves.

2. *Over the right ventricle.* In systole: A tone indicates normal state of tricuspid. A murmur indicates a relative insufficiency of the tricuspid valve; when the jugular veins pulsate there is absolute insufficiency.

In diastole: A sound indicates that it is arterial. A murmur never exists.

3. *Over the aorta.* In systole: A tone means arterial. A murmur indicates stenosis of the aortic valves or compression of the vessel.

In diastole: A sound, a normal state, a murmur indicates insufficiency of the aortic valves.

4. *Over the pulmonary artery.* In systole: A tone is the arterial sound. A murmur indicates stenosis of the trunk of the artery.

In diastole: A tone, normal state. Increased sound hyperæmia in the smaller circulation. A murmur never exists.

1. Not only the anterior aspect of the cardiac region should be examined in suspected cardiac lesions, but also the left dorsal aspect should be auscultated.

2. To determine the rhythm, the sound audible on the back from the apex-beat, or on the carotid artery should be taken as a criterion. A murmur synchronous with that pulsation is systolic, one following it is diastolic.

3. A murmur audible in a direct line upward from the apex of the heart, increasing in strength as it rises, and loudest over the mitral valve, but inaudible over the pulmonary artery, indicates *a mitral-valve lesion.*

4. A murmur audible over the apex and increasing in force toward the base, also to be heard over the aorta and on the apex of xiphoid cartilage, indicates a lesion of *the aortic valves.*

5. Murmurs extending over the whole heart, and extending all over the thorax, indicate *complications.*

Sounds existing in diseases of the blood vessels have been described in the text, in the diseases of the blood vessels.

Exploration of the Arterial Pulse.—When the tip of the finger is slightly pressed upon a superficial artery, it feels a regular shock of the vessel beneath, and is slightly raised by it. This constitutes the pulse, and takes place more or less instantaneously with the systole of the heart, and its first sound. The farther the vessel is from the heart, the less synchronous with the apex-beat (*Weitbrecht, Rechouche, Despine*). Thus the carotid artery is nearer in time with the heart-pulse than the tarsal arteries (*Hammernjk*). Since the description of blood circulation by *Harvey*, the cause of arterial pulsation was a subject of controversy. Marey is the first to clearly explain and demonstrate its causes and *modus operandi* in the several phases. Arterial tension is the product of two *distinct factors, a constant and a variable one.* When a manometer is placed into a pulsating artery the mercury is found to rise to a certain height, which varies according to the distance of the vessel from the heart; furthermore, when ventricular systole takes place, it will rise above the former level, and then fall to its normal height. This shows that during ventricular contraction there is increased arterial tension, or pressure in the arterial system; for the additional quantity of blood driven by the ventricle into the already full aorta, dilates that vessel beyond its normal caliber. The pulse is then the result of ventricular contraction and exaggerated pressure in the artery. Marey has demonstrated this fact by artificial means. The pulse must vary with the variation of either or of both factors. The older clinicists described a great variety of pulses, many of which are really of no clinical significance. *Hales* has already, in 1744, tried to demonstrate the variations of the pulse by visible means, by introducing a glass tube in the arteries of animals, and noticed the different levels to which the blood in the vessels rose. *Harrison* has constructed, in 1847, an instrument to measure that level, and called it a *sphygmometer. Chelius*, by using that instrument, has shown, in 1850, that there exists a peculiarity in the normal pulse called *dicrotism*, or of *double motion*, a fact known already to the physicians of ancient Greece. Ludwig has, by his newly-constructed *Kymographion*, added an enormous improvement to the modes of observation of variety of pulses.

In 1855 Vievordt produced a very ingenious, but very unhandy, instrument which he called a *sphygmograph* for the purpose. Ultimately *Marey* has succeeded in perfecting a very handy little instrument (*a sphygmograph*) by which both the tension of the artery and the velocity of the blood is shown and at the same time registered. In his many researches into the nature and causes of the arterial pulses and their variations, *Marey* found that, *outside the influences of the pneumogastric or the sympathetic nerves upon the action of the heart, the frequency of its pulsations is that much greater the less obstacles it meets with in the general circulation or in the discharge of its contents.* The more it meets with resistance in the vessels the more will its force increase, and the more will there be arterial tension, which will itself become the gauge of that resistance. He has formularized this fact in this manner: "*The frequency of the pulse is in inverse ratio to arterial pressure.*" By placing a manometer in the artery of a horse he has demonstrated when the animal was bled several times in succession, that the pulse became more and more frequent the more blood was withdrawn from the circulation. That the pulse was 120 with 5 mm. pressure. In man a loss of 400 grammes of blood caused much increased frequency of the pulse. *Guy* found that the pulse of an adult in an erect position is 70; sitting down, 70; lying down only 69. The same was found by *Graves.* As the heart is situated at the point of junction of the one upper and the two lower thirds of the body, the greatest number of the larger arterial trunks situated also in that region, the circulation is assisted by the arterial blood taking a downward course in the direction of its gravity. In the upper part of the body, although the blood from the heart takes a course against gravity, it is assisted by the pressure in the aorta, which tends downward. In the erect position the blood offers little resistance to the heart, and the pulse is faster, but in the recumbent position the advantage of the upright position is lost, and the heart has to increase in force, the arterial tension increases and the frequency decreases. The same is the case when the larger arterial trunks are compressed: resistance is formed to the discharge of the contents of the heart, and the pulse becomes slow, whilst dilatation of the vessels removes pressure and increases pulse-frequency. Heat, dilating the vessels, causes frequency of the pulse; cold, contracting them, produces a slower pulse. Increased muscular activity, by diminishing arterial pressure (*Marey*) from 108 to 102 mm., increases the frequency of the pulse. Rest acts reversely. Digestion increases and fasting decreases the frequency. Pregnancy sometimes increases, sometimes decreases its frequency, according to the diminished or increased arterial tension. In

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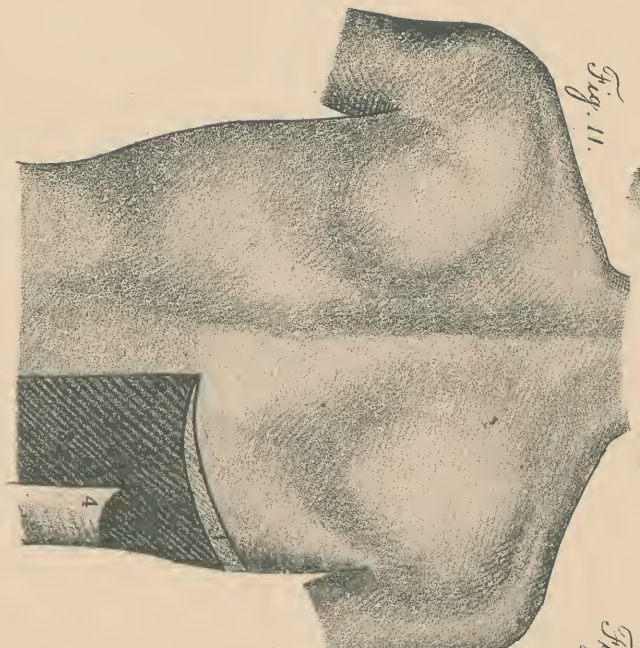

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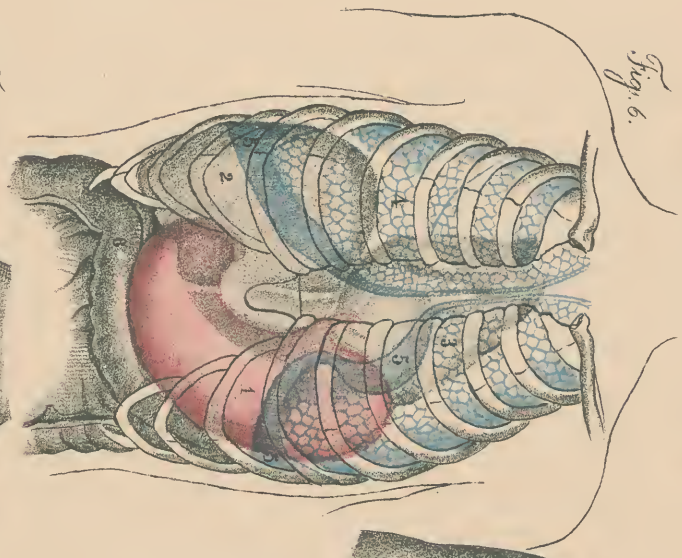

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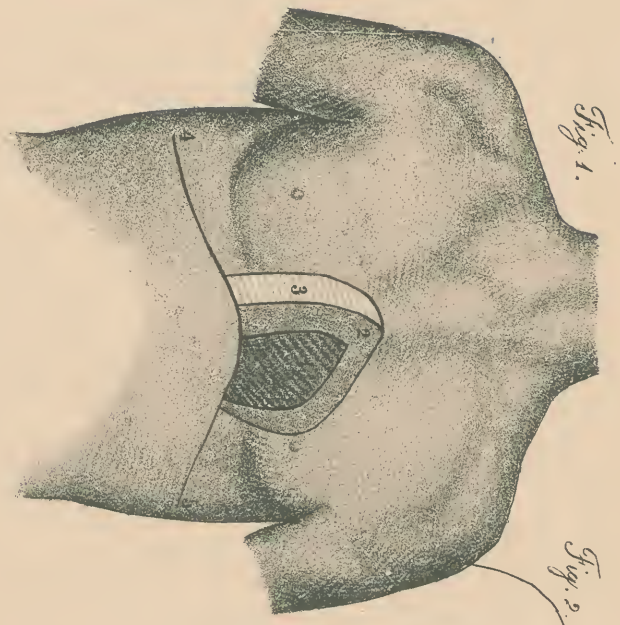

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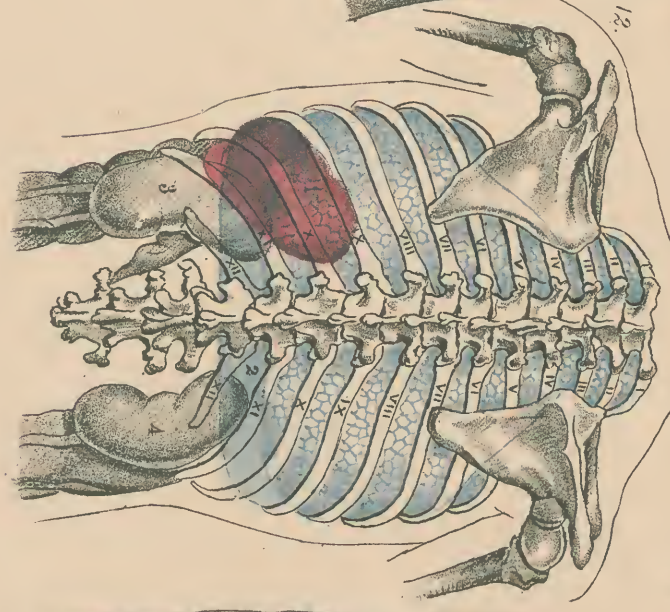

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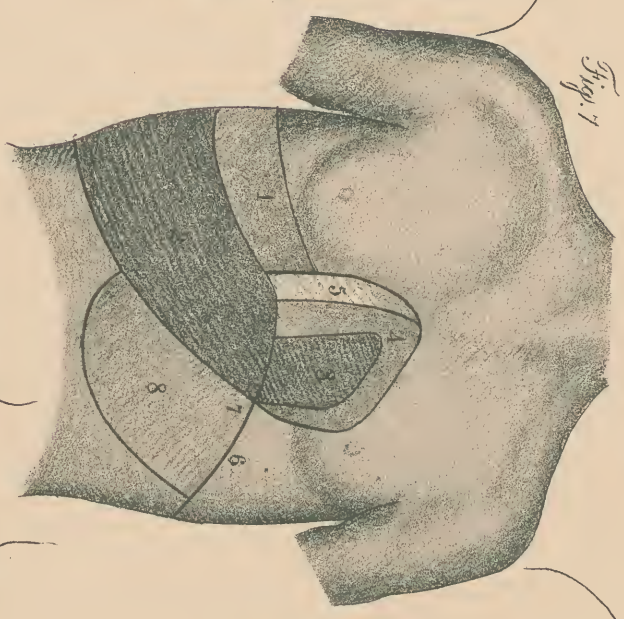

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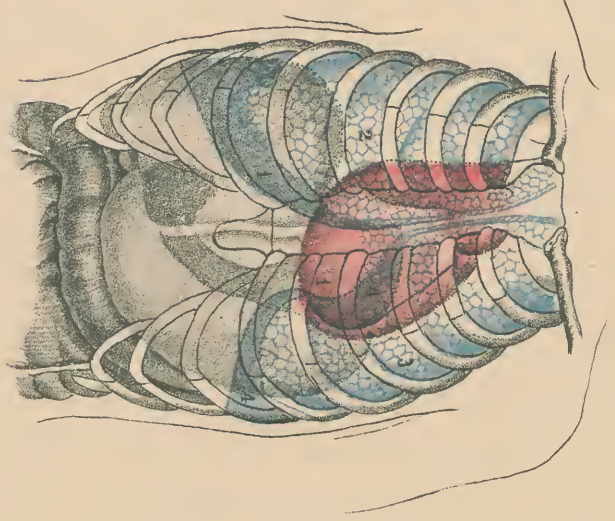

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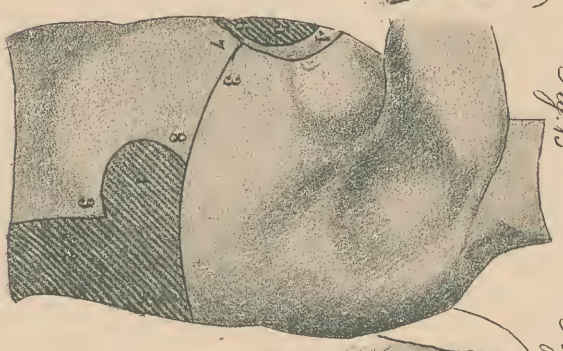

Fig. 8.

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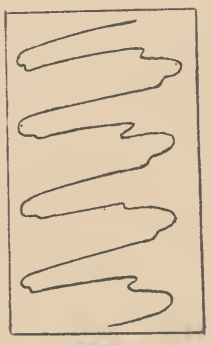

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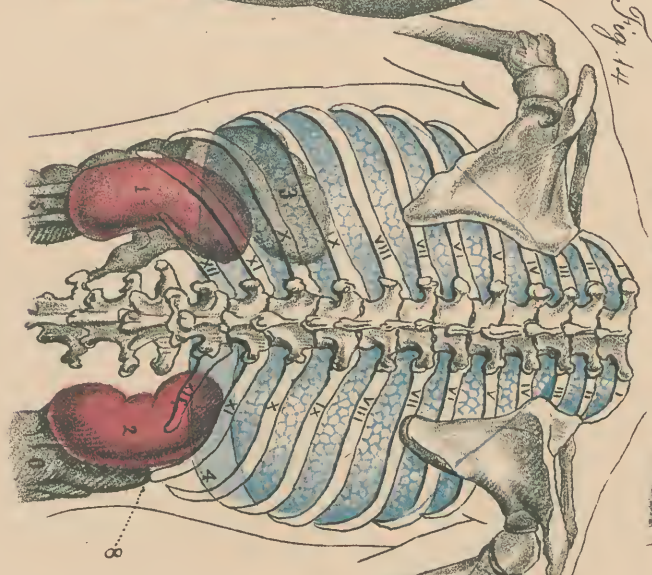

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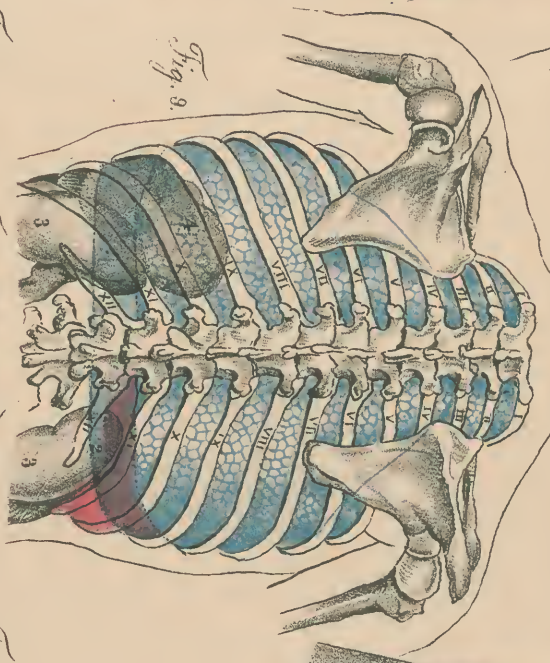

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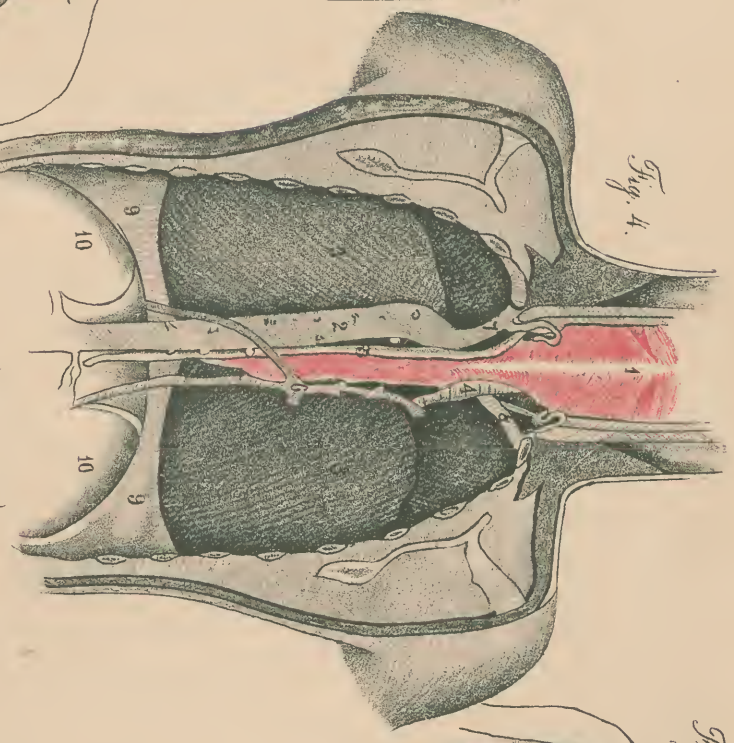

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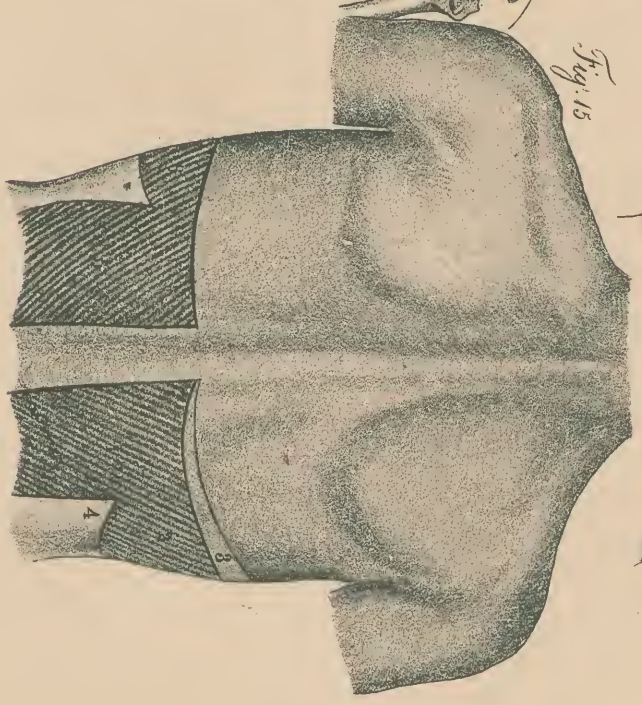

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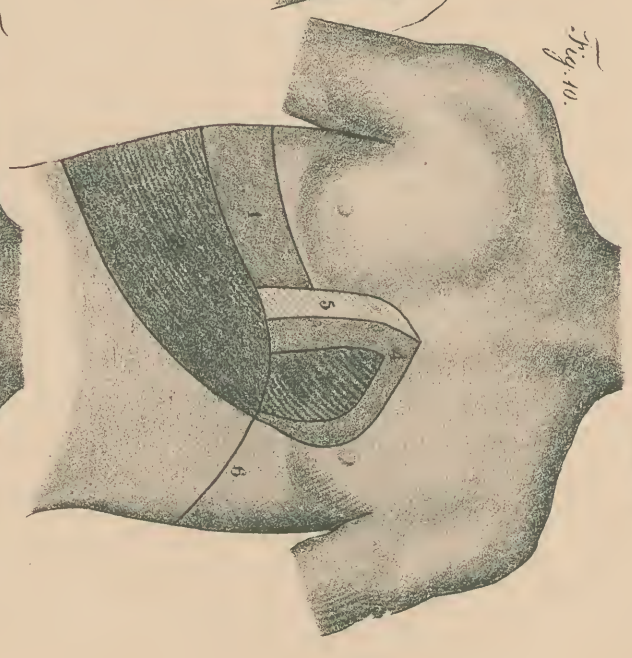

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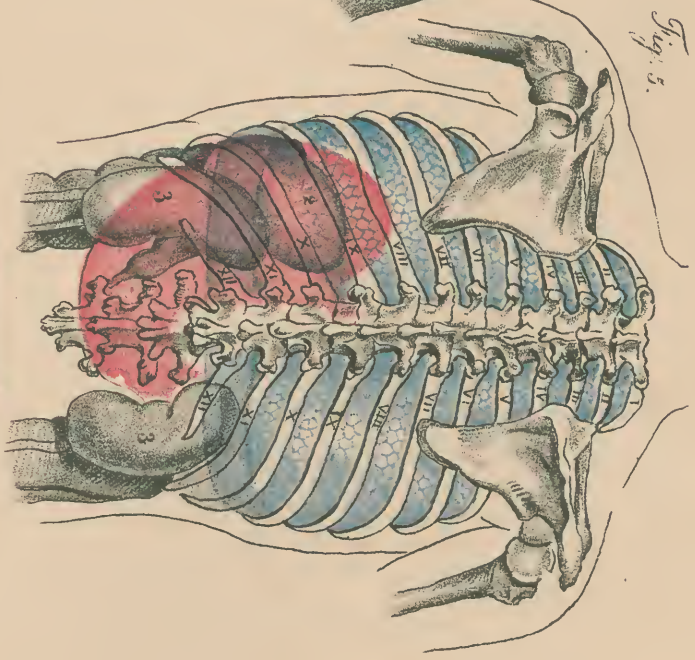

Sec. X. *Tab. I.*

MORBID HISTOLOGY.

FORMED ELEMENTS IN EXPECTORATES.

Fig. 1.

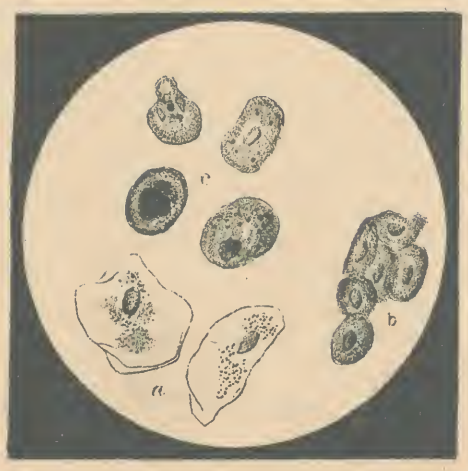

Fig. 2

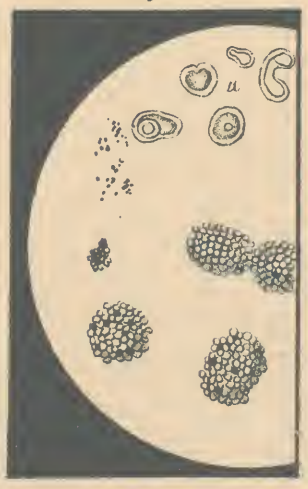

Fig. 3.

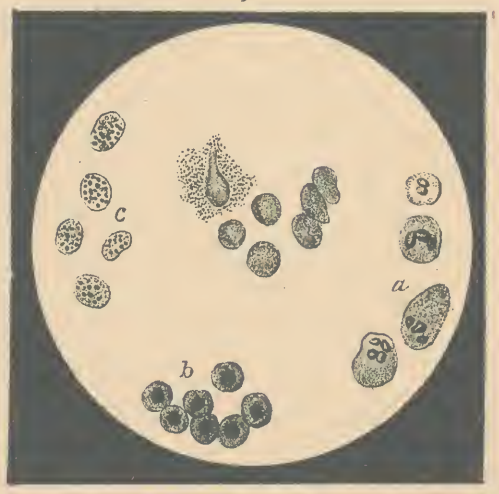

Fig. 4

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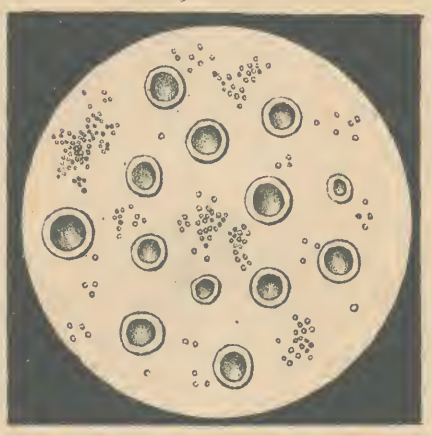
Fig. 16

Fig. 17

Fig. 18.

Fig. 19



The Donaldson Lith. Co. Cincinnati.

TABLE I.

PATHOLOGICAL HISTOLOGY.

Morphological components of expectorates.

FIG. 1.—*Epithelial cells.* (a), pavement-epithelium of the mouth, upper layer; (b), same, lower layer; (c), epithelial cells of the pulmonary alveola; (d), containing blood corpuscles and hæmatoidin crystals, in an old infarct. Fig. 2.—*Fatty degenerated epithelium.* (a), myeliniform cells, some coal-dust in the lungs. Fig. 3.—*Mucus and pus corpuscles.* (a), after treatment with acetic acid; (b), moleeules of coal-dust; (c), fatty degenerated cells. (*Eichhorst.*) Fig. 4.—*Infusoria in gangrenous sputum (monades, described by Virchow).* Fig. 5.—*Acicular margarin crystals* from the bronchi in putrid bronchitis (*Eichhorst*). Fig. 6.—*Cholesterin crystals*, in abscess of the lungs (*Leyden, Volkmann's clinical lectures*). Fig. 7.—Asthma crystals. (*Eichhorst.*) Fig. 8.—*Fibrinous coagula*, found in fibrinous pneumonia, in the bronchi, etc. (*Remak*). Fig. 9.—*Elastic fibres*, in expectorates of consumptives (*Clinics of the University of Goetting*). Fig. 10.—*Sputum in abscess of the lungs.* The elements consisting of elastic fibres, hæmatoidin crystals, and micrococci. (*Leyden, Volkmann's clinical lectures.*) Fig. 11.—*Particles of coal and coal-dust* in the expectorate of a healthy person (*Traube*); (a a), cells surrounded by coal particles. Figs. 12, 13.—*Mucous sputum.* (a), unaltered mucus-cells; (b), altered cells by treatment with acetic acid (*Eichhorst*). Fig. 14.—*Anthrax bacillus* existing in the blood of a cat infected with that disease (*Bollinger, Ziemsen's Handbuch*). Fig. 15.—*Spirillia in febris recurrens.* Fig. 16.—*Poikylocytosis.* Peculiar condition of the blood corpuscles, in a case of cancer of the stomach. Fig. 17.—*Microcytosis* in progressive pernecious anæmia (*Eichhorst*). Fig. 18.—*Cells containing blood corpuscles*, found in typhoid fever. (*Eichhorst.*) Fig. 19.—*Elementary granules*, existing in the blood of patients affected with intermittent fever (*Eichhorst*).

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genuine labor-pains the pressure is increased and the pulse is slow. *Guy Lussac, DeSaussure*, and others have shown that the more atmospheric pressure is diminished, the more frequent the pulse, whilst *Pravaz* and *Taborus* have found that the greater the atmospheric pressure the slower the pulse; and *Vivenot* found, under such circumstances, that the capillaries were contracted and offered resistance to the arterial circulation and to the emptying of the ventricle. Anæmia, tuberculosis, and fevers cause an increase of frequency, whilst meningitis, jaundice, and digitalis cause a slower pulse. In deffervescenee after severe fever the frequeney drops below the normal. The force of the pulse depends on the quantity of blood thrown into the aorta by the left ventriele as well as on the force and extent of ventricular contraetion. Force of the pulse does not always indicate the strength of the person. The greater the caliber of the vessel the more voluminous will be the pulse. In old people there is generally hypertrophy of the heart, and often a very full pulse, whieh shows high arterial tension only.

Intermittent pulse.—Irregularity in the rhythm of the movements of the heart produces irregularity of the force of the pulse. The pulse following the omitted arterial pulse will always be stronger than a normal. An obstaele to cireulation in any artery also modifies the foree of the pulse by reducing its velocity and increasing its tension in the part of the vessel above the impediment. All the above-stated faets eorroborate the correet observation of Marey in regards to the relation of the pulse to the ventrieular systole. It shows: *that the freer the arterial cavity is from impediment, the less obstacle to the entrance of the blood from the ventricle into the artery, the more speedily the successive ventricular contraction follow each other, and of course the more frequent the pulse.* After *Marey, Ducheck, Naumann, Koshlokoff*, and lately also *Franz Riegel* have done most to enrich our knowledge of sphygmography. To study a tracing or a graphic (*as the registered pulse-curves are named*), not only the whole but also every *plane* of the eurves should be closely observed. Each curve is composed of a series of undulations, traced by the rising and falling *lever* of the instrument, each corresponding to a *full act* of a pulse (see Sec. 9, Tab. VI, Figs. 1, 2, 3). In a graphic, taken from a healthy person, if a line were drawn across the summits of the eurves, it would be found to be nearly horizontal; for as the summit indicates the height, which the distended artery gives to the raised lever (which also represents the heart force), the line must necessarily remain level, for the heart retains the power and exerts a uniform pressure upon the eolumn of the blood. A line drawn through all the bases of the eurves would, in the healthy pulse, be horizontal likewise, provided the arterial elasticity be the same. The ascending line eorresponds to the arterial dilatation. Normally it is vertical, or nearly so. It assumes an oblique direction when any impediment exists in the artery to the eirculation, for the slower the elevation the more oblique is this upward moving line. The aseending line may at first follow a straight direction, but gradually a more erooked or even oblique eourse. The summit or top of the line is at the same time the apex of the triangle, formed by the junction of the ascending with the descending line.

Normally the point of the angle is aeute, for the entranee of the blood into the vessel causes a sudden drop of the arterial tension. When the vessel is rigid and not uniformally eontracted, or is inelastic, the deseending line will remain horizontal for some distance, for the arterial pressure will not be suddenly decreased, and the line will therefore remain in that position for some time. When the artery is externally eovered by much fat the pulse will also have that form. The descending line marks the gradual decrease of tension in the vessel. It is usually oblique. Its diagonal direction varies aceording to the quantity of reduction of the pressure. It may be a straight line, with a eonvex surface upward or downward, or alternate convex and concave portions. Undulations of different heights and depths may eharacterize the secondary elevations. There are three distinet elevations, the primary or eardio-aortic, the secondary first, or arterio-valvular, that is when the blood is thrown by the contracting vessel against the semi-lunar aortie valves and then again pressed into the filled vessel with less power by the valvular force. This elevation or second secondary is brought about by the second contraetion of the vessel as it is formed by its elasticity. The second or arterio-valvular shock is generally plainly felt in strong pulses by the finger. The elastic contraction is only noticed by the sphygmograph, and that not always. The cause of the second elevation was for a time a matter of dispute. *Galienus, Albers*, and *Volkman* gave it defferent interpretations. *Chelius* gave it its true definition, that is: *the rise of pressure from rebound. Vierordt* and *Marey* have confirmed this fact, and *Naumann* has shown that when there is weakening or distruction of the aortic semi-lunar valves there is no dierotism in the pulse, as the secondary elevations do not exist. As dicrotism in the pulse is due to three described factors, a disturbance in either will neeessarily modify the quality of the pulse. When the action of the heart is very weak, and the elasticity of the arteries is feeble, the pulse will be polycrotic; that is, the secondary elevations will be many but very irregular in their heights and positions, for the movement of the blood eolumn will be very uncertain and irregular. The respiratory act modifies the action of the pulse. There is aspiration of the blood from the veins into the thorax going on during inspiration. This aspiration is felt even in the capillaries, and reduced pressure in the arteries follows. The heart meets with less resistance, the pulse is quicker, and the pressural elevations lower. The reverse takes place in expiration and the pulse is modified accordingly. Ordinarily there is a eompensatory arrangement between the pressure in the arteries of the abdomen and those of the thorax, and the result is that the pulse is nearly alike all over the body; but when an obstacle to the free entrance of air into the thorax during inspiration exists, then the thoraeie vaeuum is not readily filled. Much blood is drawn from the arteries of the periphery toward the thorax and the pressure is redueed in the arteries of the extremities. On the other hand when an obstaele to free egress of air from the thorax exists the aorta becomes pressed. The blood is driven to the extremities with greater foree and the arterial tension is inereased. When the function of the diaphragm is disturbed similar eonditions exist and similar pulses are found.

Auscultation of the Air Passages and Respiratory Organs.—The respiratory organs may be eonsidered an apparatus of sound, and are made up of the following parts:

(1) *The cavity of the mouth* as an apparatus of resonance, or *a resonance-box*, which amplifies the sounds or tones passing through it from: (2) *the larynx*, whieh represents the tongue or the trompete, into which the blowing is carried on by the: (3) lungs and bronehial tubes, which represent the bellows which drive the air into the musical intrument by the inspiration and expiration. Quiet breathing takes plaee about twelve to sixteen times in a minute. In the respiratory act, certain sounds are produced: *speaking, singing, whistling*, ete. Auscultation has demonstrated that the voice which arises beyond the glottis, produees an echo within the pulmonary tissue, and that vibration is distinctly felt in the pectoral walls. The pulmonary sounds have each a different and definite signifieance and character, and serve the purpose of physieal examinations, both physiologically and in morbid states. There are inspiratory and expiratory sounds (murmurs), which differ from eaeh other as the two respiratory acts differ.

There is an inspiratory suction sound and an expirarory pressural sound, with which both thoracie resonanee and vocal fremitus are connected. The respiratory sounds not only continue in the respiratory passages upward, but also into the bronchi, aud give rise to secondary sounds passing from the bronchial tubes into the air-eontaining parenehyma of the lungs. There is then *a bronchial inspiratory* sound or murmur, and a *vesicular* sound or murmur. The latter has been named puerile respiratory murmur, because it is most distinct in the thorax of young children on account of their thin thoraeie walls; for in old age the bronchial murmur eovers the now less audible vesicular murmur. Expiratory murmur is audible over a far lesser extent of the respiratory organs, and is much more feeble. Only in morbid eonditions are these expiratory sounds increased, and may even predominate. Pectoral sounds, formed by the echo-like resonance, exist in the form of a light humming murmur, when the lungs lie elosely beneath the thoracic wall. This becomes more pronounced when the person examined is made to pronounce a few words very loud, slow, and distinct. Thoracie vibrations will then become tangible. Pectoral fremitus like all vibratory pulmonic sounds carried by solid medii, is produced by the vocal eords and carried by the solid-walled bronchi. *Bronchial murmur* is most audible over the upper portion of the posterior aspeet of the thorax, on each side of the vertebral eolumn. *Vesicular murmur*, audible nearly all over the anterior aspeet, and the lower portions of the posterior surface of the ehest.

Types of Sound in Morbid Conditions of the Organs of the Chest.—There exist: 1. Modifieation of sound when the lungs still contain air all over. 2. When some portions of the lungs contain air and some do not. In the latter there is no vesieular, but bronehial sounds. 3. When large portions of the lungs have been destroyed, or made absolutely airless. 4. When the pleural cavities are filled with air or liquid, and a distinct new sound-organ is produced in the ehest, outside of the lungs.

Pathological symptoms where the lungs still contain air may be: (1), Simple sound modifications produced by a modified mechanism of respiration. There may be (a) *dyspnœa* consisting of deep, long-drawn, yet superficial inspiration. The bronchial sound will be heard very distinetly even in the parenchyma of the lungs and will produce fremitus. (b) *Saccadated or jerking inspiration*, consisting of alternate, regular, and sudden jet-like respiratory acts (CHEYNE STOKES *respiratory type*). (c) *Weakened or suspended respiration*, when

TABLE II.

PATHOLOGICAL HISTOLOGY.

Morphological elements in urinary deposits.

FIG. 1.—Neubaur's Urometer (full size). Fig. 2.—Several forms of uric acid crystals. Fig. 3.—Uric acid crystals in the form of Rosettes. Fig. 4.—Uric acid crystals in the form of sheaves, found in certain forms of nephritis. Fig. 5.—Urate of Sodæ, in a milky sediment, found in the urin of an epileptic person. Fig. 6.—Urate of Sodæ in fan-shaped agglomerations of crystals (*Ulzmann* and *Hoffmann*). Fig. 7.—Uric acid crystals in a sediment formed by addition of acetic acid to the urin. Fig. 8.—Several forms of urate of ammoniæ. Fig. 9.—Ammonia-phosphate of magnesia, found in alkaline human urin (*Terphosphate*). Fig. 10.—Crystals of neutral phosphates (*Ulzmann* and *Hoffmann*). Fig. 11.—Crystals of phosphate of magnesia (*Stein, deutsh. arch. f. Klin. Med. Vol. XVIII*, 1876). Fig. 12.—Crystals of carbonate of lime; (a), in the urine of a rabbit; (b), a rare case of the kind in the human urine (*Golding Bird*). Fig. 13.—Crystals of oxalate of ammonia in the human urin; (a), prismatic; (b), dumb-bell-shaped crystals. Fig. 14.—(a b), hippuric acid crystals in the urin of a rabbit after præcipitation with dilute nitric acid (*Eichhorst*). Fig. 15.—Cystin crystals in human urine (*Ebstein, deutsh. arch. f. Clin. Med., Vol. XXIII, pg.* 139). Fig. 16.—Leucin, styrosin crystals in acute yellow atrophy of the liver (*Ferrichs*). Fig. 17.—Xantin crystals in urinary deposits (*Bence Jones, Journ. Clinic Society, London, Vol. XV.*, 1862). Fig. 18.—Epithelial cells in urinary sediments; (a), epithelium of the bladder, upper layer; (b), the same in the middle and deepest layer; (c), epithelial cells of the uriniferous tubules (*Eichhorst*). Figs. 19, 20.—(19), epithelial tubules; (20), epithelial cylinders in urinary sediments, in acute nephritis. Fig. 21.—Cylinders of blood corpuscles found in a case of acute nephritis (*Eichhorst*). Fig. 22.—Hyaline, cylinders, in urinary sediment of a cat; after hypodermatic injections with chromic acid and producing chronic parenchymatous nephritis. Fig. 23.—(a), finely granular urinary cylinders; (b), coarsely granular cylinders in urin of the cat affected with chronic nephritis (*Eichhorst*). Fig. 24.—Cylinders in amyloid degeneration of the kidneys (*Eichhorst*). Fig. 25.—Urinary cylinders with deposits of (a) fat molecules; (b), oxalate of lime crystals; (c), red blood corpuseles; (d), epithelial cells of the uriniferous tubules (*Eichhorst*). Fig. 26.—Sarcina Ventriculi, found in the stomach.

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the external respiratory apparatus is paralyzed, or when great obstacles are placed in the way of the entering air. In the latter case cessation of vesicular murmur will be a sure diagnostic sign. (d) *Cardio-pulmonic murmur* is produced when the apex-beat interferes with the inspiratory act, and the breathing has a rhythmic character. (e) *Vesicular rattling.*—When the vesicles are partly filled with mucus.

Harsh respiratory sounds are either temporary or permanent. They may be caused by morbid conditions of the respiratory passages (pharynx or larynx), such as whistling, hissing, snoreing, gurgling, rattling sound, etc.

Accentuated, rough respiratory sounds.—The normal pluerile or sonorous respiratory sound of children indicates in adults a condition of an acute pulmonic disease, the circumstances being dyspnœa and diminished vesicular elasticity.

Expiratory contractions cause long-drawn expiratory sounds. The air to be breathed out accumulating in the air passages can not at once escape and requires an effort on the part of the expiratory muscles to accomplish it. The sound produced is necessarily longer drawn and modified. This condition may be temporary or permanent. When the obstacle exists in the bronchi there will be bronchial *expiratory murmur* or sound, which will accompany *the whole respiratory act*, with intense *bronchial reverberatory* sound from the increased force of the vibrations. *Rhonchi* or *rattles*, *rales*, are produced when liquids are contained in the respiratory passages or organs, together with air. Rattling sounds of respiration is typified by rattling of mucus in the respiratory passages. The liquids interfere by their vibrations with those of the air and thereby modify the sounds produced by it. The changes of sound phenomena under these circumstances depend on the quantity and quality of the liquid by the side of the air.

Rattle or rales are empirically divided in moist and dry, the first signifying that the liquid—whatever that be—is thin or flowing readily, so that its forward and backward movement in the tube or cavity is easily effected, whilst by a dry rattle or rale is meant that the liquid is more or less inspissated and not so readily moved to and fro, and hence the sound is apparently different in the motion of the one than in that of the other. The same is the case with *large vesicular* rale and *small vesicular*. In this the sounds appear as if they were produced by the bursting of larger or smaller bubbles or vesicles, the report being in a lower note when large bubbles burst than when smaller ones break. Very many modifications of rattling sounds or rales are clinically observed, most of which being of very subordinate importance. As nearly all forms of rale are due to collections of liquid in the respiratory organs, of course catarrhal conditions, both chronic and acute, of the mucous surface of those organs will be foremost in production of such classes of changes of the respiratory sounds. Next to these conditions œdema and hæmoptysis, by causing collection of liquid in those cavities, are liable to give rise to rales. A number of *rhonchi* are heard when the air cells are much dilated, when their walls are much thickened. They are the so-called vesicular crepitations, produced by variation of sound of the vibrating air in the larger or smaller cells, and the greater or lesser tension of their walls.

2. *Pathological sounds of airless parenchyma of the lungs.*—When the tissues have become solid, the sounds which normally exist in the presence of air in the cells are lost and are replaced by a new order of sounds. Vesicular respiration does not exist and the bronchial sounds pass into that of the thorax so that the respiratory sounds have the same character on the thorax as normally over the trachea. In extensive and great solidification of the parenchyma the bronchi become rigid and the conductibility of the solid tissue propogates the sounds into the thoracic wall and produces consonance. The register of such sound is: (1), *Bronchial sound*, greatly increased resonance, by the solid tissue and chest wall, of the respiratory sound. (2), *Bronchial rale*, widely differing from such as exist in air-containing lung tissue. (3), *Bronchophony.*—The bronchial sound is clear of the otherwise only muffled thoracic resonance. It appears as if the examined person was speaking directly into the listeners ear. Vocal tremor is often stronger, sometimes weaker than normal.

3. *Pathological sound modification when portions of the lung tissue are destroyed.*—Cavities and obliterated portions of the lungs, from whatever cause, when they contain air will yield quite different sounds than when a portion is filled with liquid or solid substances, or when the walls of such cavities are solid, emphysematous, or simply filled with air or liquid. The extent of such cavities are also an important factor in modifying the sound phenomena, for in all the mentioned circumstances the unequal distribution of medii necessarily produce the greatest variation of sound.

4. *Pathological changes of sound in hydro-thorax and pneumo-thorax. In both there is amphoric sound*, that is, a kind of echo or reverberatory tone, readily produced by striking upon the walls of a pitcher or a jug, indifferently whether the vessel partly contains liquid or not. The amphoric sound is a higher grade of resonance and has all the characters of a complex echo. The regularity of its reverberatory projections causes it to assume a character of a tone, the pitch of which is so much higher the narrower the space where the echo is evolved.

Friction sound.—There are a number of sounds which are produced by friction resulting from uneven surfaces moving upon one another. Friction sound, long known in surgery as the peculiar sound produced by uneven surfaces of bone upon one another, has been observed by Reynaud, in 1819, to exist in either of the three serous sacs of the thoracic and abdominal cavities. In placing the left palm flatly upon the ear a friction sound will be heard, when a finger of the right hand is passed over the metacarpal bones of the left hand. When either of the surfaces of the serous sacs facing each other become uneven or rough, the movement of the viscera they enclose will cause those surfaces to move upon one another and friction sound will be produced. Endo-cardial friction sound follows the rhythmic movements of the diastole and systole. The pericardial are not so rhythmic.

Topographic anatomy of the abdominal and pelvic organs.—The abdominal organs proper situated below the diaphragm are: the stomach, the intestinal canal, the liver, pancreas and spleen. The walls of the abdominal cavity are partly osseous, muscular, and connective tissue. The lower ribs, the lumbar portion of the vertebral column, and the ossa innominata form the bone-structure; the diaphragm, the internal and external oblique, the rectus abdominalis, the transverse abdominis, the pyramidal and quadratus lumborum muscles make up the fleshy portions of the wall; and the fasciae make up the membraneous closures of that cavity. Internally it is lined by a membrane, which stands in relation to the abdominal viscera and the parieties of the cavity as the pleura to those of the chest. For convenience of study the abdominal cavity has long ago been conventionally divided in zones or regions, which are separated from each other by imaginary lines drawn horizontally from side to side. The first horizontal line is supposed to be drawn from the lower border of the twelfth rib on the right side to a similar point of its fellow on the left; the second line is drawn from one anterior superior spinous process of the ilium to the other opposite. Above the first line lies the epigastric region; between the two the meso-gastric, below the lower the *hypochondriac* region. All three horizontal lines are supposed to be traversed by three vertical lines parallel to each other. To the right and the left of the outer lines are the right and the left *hypochondriac regions*, in the *epigastric zone*. Between the two is the *gastric* region, in the same zone. In the meso-gastric zone there are the *umbilical* and the *two lumbar* regions. In the hypogastric zone are the iliac and pubic regions.

Contents of the abdominal cavity.—The stomach is chiefly situated in the left hypochondriac, the epigastric, and a small portion in the right hypochondriac regions. Farthest to the left the *fundus* is situated still beneath the ribs, and passes to the right with its cardiac portion, which is situated more in the epigastric region, whilst its pyloric extremity extends into the right hypochondriac region. The fundus touches above the diaphragm; to the left and posteriorly, the spleen, with which it is connected by the gastro-splenic ligament.

The cardiac extremity lies immediately at the foramen œsophageum of the diaphragm. The pyloric extremity extends quite beneath the liver to the *quadrate* lobe. The upper surface of the stomach is bound posteriorly and the right by the posterior surface of the liver, to the left by the anterior wall of the abdomen. The posterior surface lies in front of the large blood vessels and the pancreas. The lesser curvature is turned toward the liver, the greater curvature faces forward and downward, touching the transverse colon, to which it is attached by the gastro-colic ligament. At the pyloric extremity begins the intestinal canal. This is divided into the large and small intestines. The duodenum, the first of the small intestines, has no convolutions, is situated against the posterior abdominal wall, and winds round the head of the pancreas. In its course, the superior horizontal part runs beneath and behind the liver, having the quadrate lobe of the liver and the gall bladder in front; the descending part is limited on the right and behind by the inner border of the right kidney, to the left and behind by the inferior vena cava, downward by the psoas muscle. In front is the lower part of the right lobe of the liver, and the right colic flexure. The inferior horizontal part lies, besides in front of the above-named

MORBID HISTOLOGY. **Sec. X.** *Tab. II.*

FORMED ELEMENTS IN URINARY DEPOSITS.

Fig. 26.

Fig. 1.

Fig. 2.

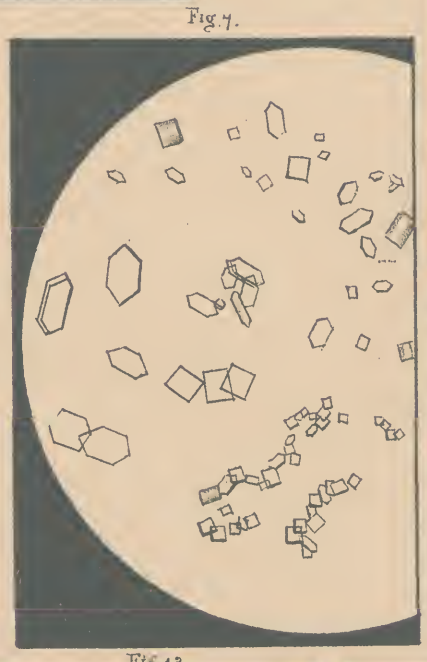
Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

Fig. 7.



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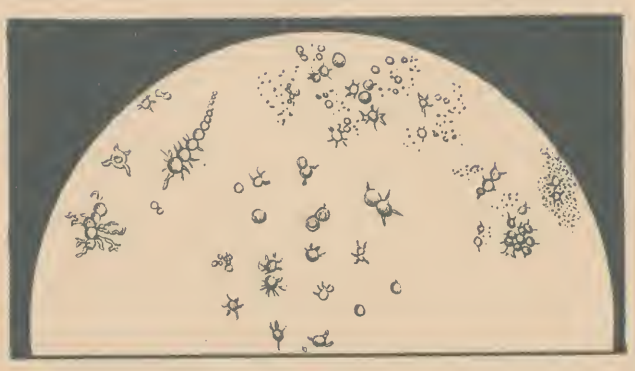

Fig. 9

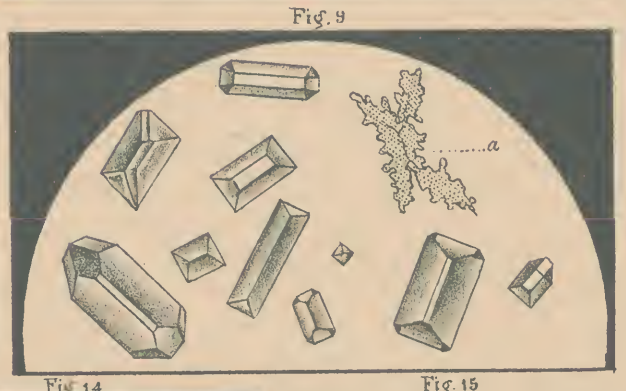

Fig. 10

Fig. 11

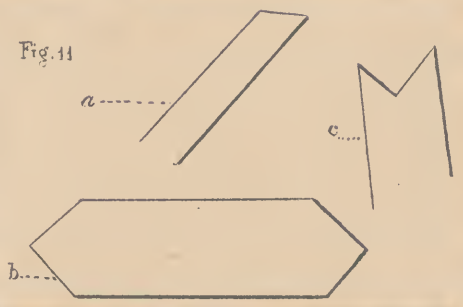

Fig. 12

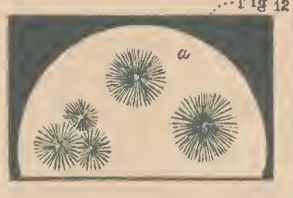

Fig. 13

Fig. 14

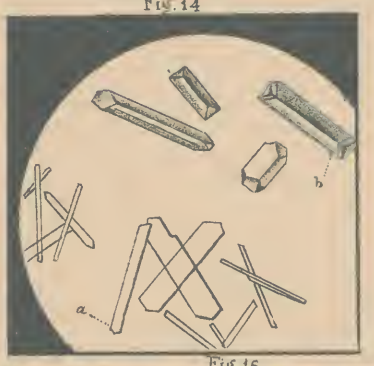

Fig. 15

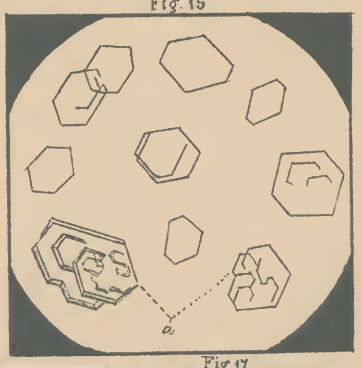

Fig. 16

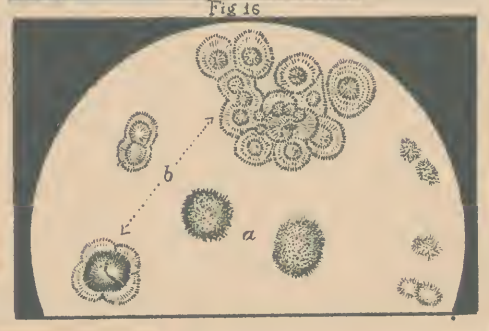

Fig 17

Fig. 18.

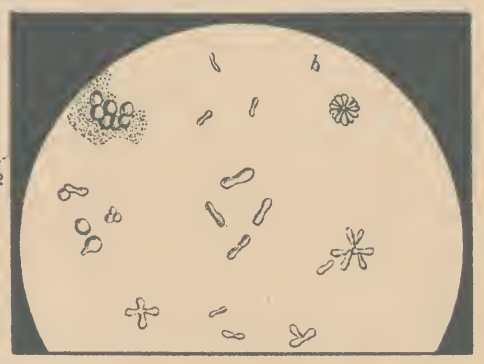

Fig 19

Fig. 20.

Fig. 21

Fig. 22.

Fig. 23.

Fig. 24

Fig. 25.

The Donaldson Lith. Co. Cincinnati.

Sec. X. *Tab. III.*

CHANGES OF FORMED ELEMENTS OF BLOOD.

BLOOD CORPUSCLES AND HAEMATOBLASTS.

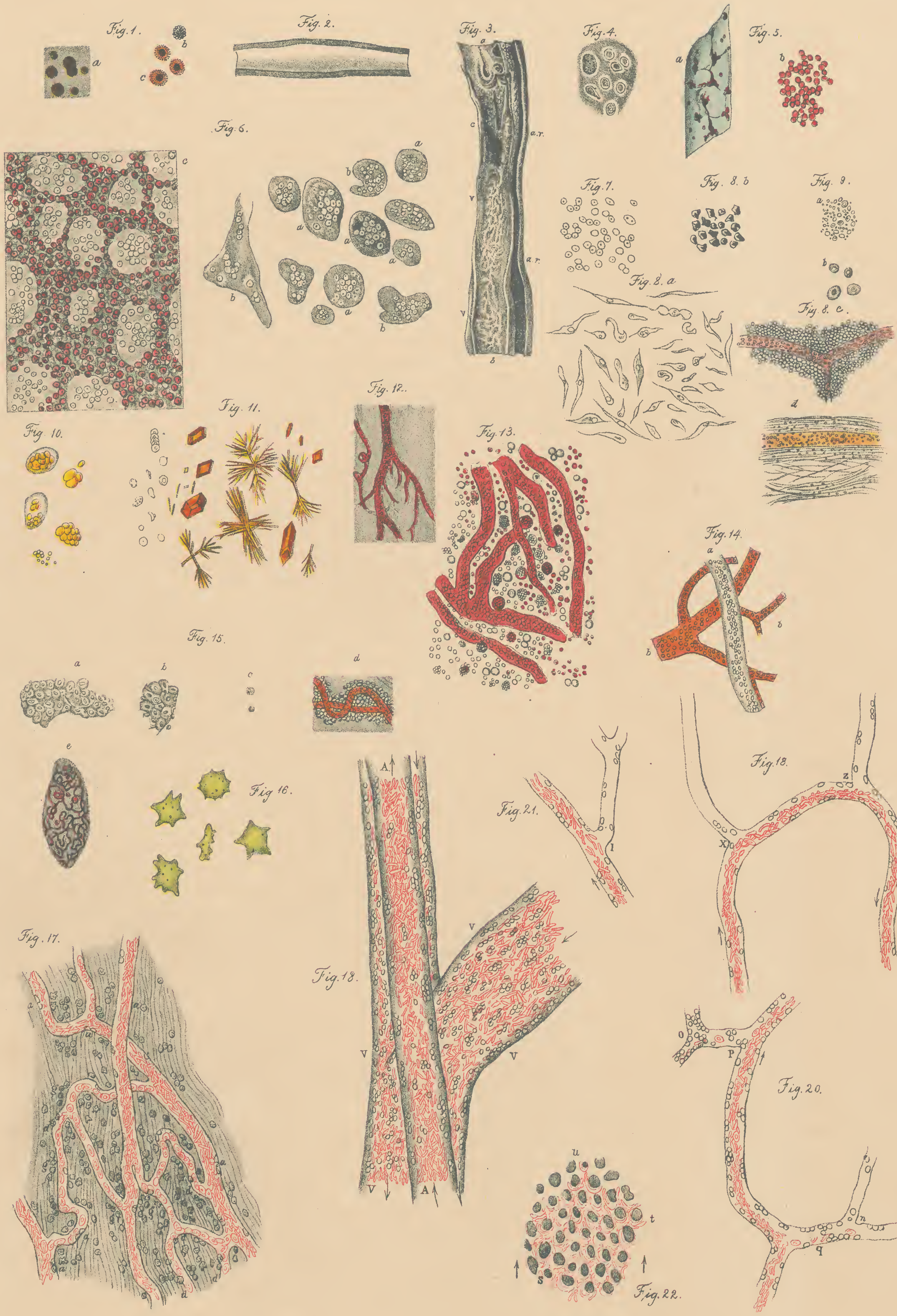

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TABLE III.

Metamorphosis of the formed elements of blood.

FIG. 1.—*Blood corpuscles and hæmatoblasts.* (a), blood corpuscles undergoing granular change; (b), colorless blood cells; (c), hæmatoblasts. All derived from a blood-coagulum in the femoral artery of a case of gangrene of the foot. Fig. 2.—A dilated capillary, from the mesentary of a frog. Fig. 3.—Coagulated blood, pus, and fibrinous efflorescences on the inner surface of a vein, in endophlebitis. (a), venous valves. The outer tunic of the vein adheres to the artery (ar); (b b), extremity of the vein (v v) free from inflammation. Inner coat of vein is rough (c). Fig. 4.—Mucus corpuscles filled with fat particles and nuclei in a state of developing epithelium. Fig. 5.—Developing blood corpuscles (*hæmatoblasts*), taken from an enchondroma. (a), thin layer of the tumor; (b), hæmatoblasts taken from that plate. Fig. 6.—Hæmatoblasts enclosed in cells, in a state of development. (a), cells without processes; (b), large cells resembling *giant cells;* (c), hæmatoblasts with apparent nuclei, some are colored, some colorless. Fig. 7.—Spheroid cells containing nuclei. Fig. 8.—(a), connective tissue cells in a state of transformation, in a pseudo-membraneous exudate; (b), epithelium infiltrated with lime and disfigured; (c), granulation in a healing wound; (d), a pseudo-membrane, showing newly developing vessels and surrounding connective tissue. Fig. 9.—Regeneration of the epithelium of the skin after abrasion of the epidermis and cutis by a fly blister. (a), *nucleus* and *nucleoli;* (b), perfectly developed cells. Fig. 10.—Blood corpuscles of different sizes. Fig. 11.—Hæmatoidin crystals and destroyed blood corpuscles. Fig. 12.—Vascular condition in hyperæmia of the renal pelvis, in *pyelo-nephritis.* Fig. 13.—Congestive condition of the *intrauterine* surface after child-birth. Colorless and colored corpuscles are mixed with fat globules and detritus of the epithelial and blood elements in the tissues. Fig. 14.—Hyperæmic condition of the *pia-mater.* The lymph vessels (b) are colorless, the blood capillaries are filled with blood and are very red (a). Fig. 15.—Processes of granulation in wounds healing by second intention. (a), rudimentary state of the newly forming epithelial cells; (b), the developed cells; (c), pus corpuscles; (d), granulations, consisting of blood vessels and spheroid cells. A number of pale hæmatoblasts are readily seen; (e), a less magnified view of a granulating mass in a wound. Great quantities of vascular loops lie imbeded in newly forming tissue. Fig. 16.—Colored blood corpuscles containing *monades*, with irregular outlines. Fig. 17.—Inflammatory processes according to CONHEIM. The microscopic figures from 17 to 22 are from drawings from nature, by Prof. Sommer, at the anatomical institute of Greifswald (*Hueter's Allgem. Chirurgie*). Fig. 17. Twenty-four hours after setting in of inflammation there is a tendency in some portions of the blood vessels (a a) that the colorless corpuscles move along their inner walls and become stationary (d d), then to diapedesis or exit of the cells from the interior of the vessels by first protruding a number of processes through the vascular wall. They at first remain on the outer wall, but afterward enter into the tissues (g g) as infiltrates. Figs. 18 to 21.—Fever processes artificially induced by Hueter and Greveler. (a a), artery; (v v), vein. The arrows indicate the direction of the current of the blood. Fig. 18 shows the colorless corpuscles forming groups and passing to the walls along which they slowly move. Fig. 19 shows some vessels which are nearly impermeable to colored corpuscles by the occlusion produced by the colorless. At the place of division into branches (x z) a few colored cells pass into the capillary. Fig. 20, at (p), is seen how the colorless cells choke up the cavity of the vessels. (o) is a portion of the vessel completely filled with colorless cells, whilst at (P) it is only partly so. At (N) the capillary is nearly empty. Fig. 21 at (l) the vessel is equally empty. Fig. 27 shows the peculiar form which the colored corpuscles assume in the lungs of a frog in inflammation. (S T U) show their tortuous movement.

parts, also in its passage into the jejunum, in front of the body of the pancreas. In front it is covered by the transverse colon and is here but scantily covered by the peritoneum. Into the inferior and interior wall of the descending portion empty the pancreatic and hepatic ducts. At the left side of the third lumbar vertebra the duodenum, after passing through the transverse mesocolon, passes into the jejunum. From this intestinal division begin the intestinal convolutions which are attached to the mesenteric folds and float freely in the abdominal cavity so that the upper and middle part of that cavity is occupied by the jejunum, the lower division of the abdomen and upper division of the pelvic cavity by the ileum. The lower division of the ileum ascends from the pelvic cavity upward and to the right and passes into the inner surface of the large intestine, the coecum, in the right iliac fossa. The coecum with its ileocoecal valves are situated in the described fossa upon the right iliacus muscle. The coecum passing into the colon rises upward as the ascending portion of the intestine, in front of the right posterior abdominal wall and in front of the right kidney to the lower surface of the right lobe of the liver, and is covered in front by some intestinal convolutions. It bends here sharply to the left and slightly downward, forming the *right colic flexure*, forming a downward tending arch transversely, below the great curvature of the stomach, to the left, down to the front of the lower division of the spleen, as the *transverse colon*. Posterior to it are the horizontal portions of the duodenum and the pancreas. In the left lumbar region the colon bends again downward and backward, forming the left flexure, and descends to the broad expanse of the left iliac bone as the descending colon. It now turns in a sharp curve to the right and downward, descending to the front and below the promontorium, receiving the name of *sigmoid* flexure, and here passes into the rectum.

The liver occupies chiefly the right hypochondriac region, but extends into the left hypochondriac and the epigastric regions, and lies in the right concavity of the diaphragm, its obtuse border slightly facing upward and backward, its sharp border forward and downward, slightly protruding below the lower borders of the ribs. The posterior surface of the right lobe is in front of the upper part of the right kidney, the right colic flexure, the commencement of the transverse colon, and the descending portion of the duodenum. The spigelian lobe lies upon the lumbar portion of the diaphragm and the lobus quadratus is upon the superior horizontal part of the duodenum. The left lobe is in front of the pyloric part of the stomach, the small curvature and the lesser omentum. The gall bladder usually protrudes from beneath the anterior border of the liver. The pancreas lies transversely across the vertebral column, the aorta and vena cava, extending from the descending part of the duodenum to the spleen. The head is mostly to the right, its body to the left. In front of the pancreas is a portion of the stomach and the transverse colon. The spleen is situated in the depth of the left hypochondriac region. Its concave surface lies against the fundus of the stomach and tail of the pancreas, its convex surface against the concave surface of the diaphragm in the place of the ninth and eleventh ribs, to the left and outward. Inferiorly it rests upon a fold of peritoneum, the pleuro-colic ligament. In front it touches the right colic flexure, behind, the left kidney and supra-renal capsule. The kidneys lie on the posterior abdominal wall on each side of the vertebral column, in the lumbar region, upon the quadratus lumbarum muscles, and their upper parts upon the lumbar portion of the diaphragm. Their external border is bounded by the lateral abdominal walls, the inner border by the first to the third lumbar vertebræ. In front, the right kidney is covered by the descending portion of the duodenum, the ascending colon and the right lobe of the liver; the left has in front the desending colon, the spleen and the tail of the psoas. The ureters spring from the hilum of each kidney and descend on each side over the inner division of the quadratus lumbarum and the psoas muscles, downward and inward, across the iliac vessels into the pelvic cavity, and along its posterior wall, near the rectum, to the fundus of the bladder, into the walls of which they enter. The bladder is situated immediately behind the pubic symphysis. Its upper border projects, when the organ is empty, above the upper borders of the pubis. When filled it may project high up in the abdomen. Posteriorly it lies, when empty, in the pelvic space and is limited, in the male, only by convolutions of the smaller intestines, in the female, also by the uterus. The full bladder touches the rectum behind.

Normal and abnormal sounds of the abdominal and pelvic organs.—The liver yields by percussion a dull non-tympanitic sound where it is very thick; a dull but slightly tympanitic sound where it is thin and when it covers the clear-sounding bowels. The area of its dull sound varies in extent even in healthy persons. In diseases it may extend and contract its boundaries enormously. During respiration it shifts upward and downward with the movement of the diaphragm, the upper and lower viscera.

Percussion of the spleen.—As this organ very often undergoes great change of volume, the area of dull sound proper to it is often covered by other sounds, such as that of the liver, the kidneys, or the stomach. In enlargement of the spleen its dull sound may often extend deep down into the abdomen.

The pancreas, even when greatly enlarged, can not be diagnosed by percussion from its peculiar position between the intestines.

The kidneys usually yield a dull sound on percussion, yet their size is little fit to give a clear idea, unless very much enlarged or extremely or very much reduced, of their true condition by percutory examination only. Especially is the lumbar region but little fit to yield satisfactory sound symptoms. Palpation especially, considerable pressure in the renal region, will sooner show a resisting hard enlarged kidney.

The stomach and the intestines being hollow organs and covered mostly by more or less elastic walls are most fit for percutory diagnosis. When either of those intestines are distended with gases, and the abdominal walls are not tightly stretched, then there will be nearly a uniform loud and clear drum-like sound. Very seldom will metalic consonance be produced on percussion. But when the abdominal walls are tightly stretched over the distended intestines, the sound produced by percussion will not be so loud nor tympanitic, nearly all over the abdomen. Liquids collected in the abdominal cavity, but not in special *sacs* (cysts), will flow toward the lowest level in the cavity when the patient changes his place, for the gases contained in the intestines will tend to occupy the higher levels in that cavity and the intestine will float on the surface of the liquid. The places occupied by the liquid yield a dull sound when behind the liquid there is air-containing intestine. In diffuse peritonitis, when the intestines are distended by much gas and the abdominal walls are tensly drawn over them, there will be a non-tympanitic muffled sound on percussion, though the exudate may be plastic and of insignificant thickness. When the intestines are not distended with gases, then there will be a clear sound unless there be massive exudation between the membraneous folds. Adhesion of the intestines, or synechia of all with the abdominal walls, causes tumefaction of the abdomen when there is much gas in the bowels, Percussion sound under such circumstances will be, nearly all over the abdomen, loud but not as clear as normal. Tumors or other solid bodies in the bowels or abdomen, unless of a good size, will not yield sufficiently distinct sounds. Paralysis of the muscular coats and morbid alterations of the mucous membranes of the bowels often modify the normal intestinal sound by collection of gases in those cavities. Large tumors of course greatly interfere with the normal intestinal sounds. The uterus on account of its deep position in the pelvic cavity and the normal ovaries from their small size give no sound on percussion. Enlargement of either of those organs will produce a dull sound by percussion when they are situated beneath the abdominal walls. When the bladder is distended and projects high above the pubis it will give a dull percussion

TABLE IV.

Morbid histology of abdominal typhoid, scarlet fever, tuberculosis, gangrene.

FIG. 1.—*First stage of abdominal typhoid.* Exudate in the plaques and solitary follicles of the ileum. (a), surface of the mucus membrane, vascular condition of the patches and solitary follicles; (b), transverse section of the same showing its enlarged state. Figs. 2, 3. —*Second stage of typhoid fever.* Exfoliation of the epithelium and disappearance of the exudate. The plaques are surrounded by injected tissue, the glands and follicles are bare of epithelium. Fig. 2, natural size; fig. 3, much magnified. Figs. 4, 5.—*Total disappearance of exudate.* A portion of the mucous membrane left uninjured (natural size). Figs. 6, 7.—*A month after cessation of the fever process.* The plaques are obliterated and their places are indicated by dark spots upon the intestinal surface. Fig. 6, the epithelium is exfoliated, natural size; fig. 7, slightly magnified. Fig. 8.—Spheroid cells from the cellular infiltrate in the follicles. Fig. 9.—*Anthrax.* (a), pus from an abscess of the skin; (b), the postule, natural size; (c), pus in a vein; (d), pus in the lungs. Figs. 10–14.—*Intestinal changes in scarlet fever and desquamative process in the kidneys.* Fig. 10.—Wrinkled surface of Peyer's plaques. The follicles are broken and collapsed. There was no exudate. Near the ileo-coecal valves the mucous membrane readily exfoliates. Fig. 11.—*Solitary follicles of the colon*, thickened and plainly visible (natural size). Fig. 12.—A broken and collapsed follicle surrounded by Lieberkuhn's glands (magnified). Fig. 13.—The same, more magnified. Fig. 14.—Uriniferous tubules with much enlarged epithelial cells and nuclei. Fig. 15.—Chyliferous vessels filled with tubercular masses. Fig. 16. —Enlarged and dilated capillaries in the lung tissue in tuberculosis, imitating ordinary fibrous pneumonia. Fig. 17.—Gangrenous slough in a cartilage. The fibres are separated from each other. Fig. 18.—*Condition of the intestines in cholera.* A portion of the ileum with swollen plaques. Fig. 19.—Solitary follicles. Fig. 20. —Broken down follicles in the colon. Fig. 21.—Vascular condition of the mucous membrane of the intestine, at the follicles, in a case of hæmorrhagic *lepto-meningitis.* Fig. 22.—Epithelium of the bladder in cholera. Fig. 23. Corrugated appearance of a plaque in cholera. Complete denudation of the epithelium, natural size. Fig. 24.—*Epithelium of the uriniferous tubules in cholera.*—The kidneys were in an intense state of catarrh. Fig. 25.—Uriniferous tubules in the cortical portion of the kidney. (a), malpighian body with obliterated glomerulus. Fig. 26.—A villus of the intestine denuded of its epithelium. Fig. 27.—A portion of the same intestine. Fig. 28.—A much magnified view of a small group of Lieberkuhn's glands denuded of their epithelium. Fig. 29.—A portion of the colon showing the glands without epithelium but filled with spheroid cells. Fig. 30.—Formed elements of the intestinal mucous membrane, found in the stools and vomited masses in cholera. Fig. 31.—Granules and cylindrical epithelium of the exfoliated mucous membrane. Fig. 32.—A villus bare of its epithelial covering. Fig. 33.—Granular nuclei of exfoliated epithelium in cholera typhoid. Fig. 34.—Floccular masses in the stomach of cholera patients, consisting of destroyed epithelium. Fig. 35.—Lymph cells in the lymphatics mixed with blood in cholera. Fig. 36.—Fibrous structure and detritus in the destroyed membranes of the intestines. Fig. 37.—Billiary pigment and albuminoid substance found in the bile of cholera patients. Fig. 38.—Morphological elements, found in the sediments of cholera stools. Fig. 39.—Rice-water stools, in the first attack of cholera. Fig. 40.—Formed elements in ejecta of gangrenous enteritis. Fig. 41.—Normal uriniferous tubule of an infant two days old. Fig. 42.—Destroyed epithelium in nephritis in infants. Fig. 43.—Uninary cylinders in nephritis of infants. (The figures in this plate were drawn from nature and the microscope by Dr. Vandendale. *Gluge. Atlas d. Patholog. Anatomie*).

sound. The same will be the case when its walls are very much thickened and enlarged. Under this circumstance it will be more resistant than when only distended by liquids.

Clinical thermometry.—The use of the thermometer in diseases is to ascertain the degree of temperature of the body of the patient. It furnishes a very correct measure of the intensity of the morbid process, and well-defined notions of either the constancy or fluctuations of its course. It assists also in formulizing the general symptomatic phenomena of diseases and often to predicate their probable terminations (Prognosis). The kind of instrument to be used may be said to depend on individual taste and national standard. Both the standard of Fahrenheit Celsius and Reaumur are in use. Thermo-electric instruments are also occasionally employed, but this only for finer investigations of physiology, etc. The self-registering instrument is certainly the most preferable for ordinary examinations in the daily practice, and a number of such instruments are sufficiently supplied by the trade. The arm-pit is oftenest used where to place the instrument. Rarely the Rectum or the Vagina. Before placing the instrument in the axilla, it is recommended by Liebermeister to wipe off all the sweat that usually exists in that cavity, then after placing it to close it up, by placing the arm against the body and forearm upon the chest. At least ten minutes, better fifteen, should be given the instrument to stay against the body before its removal for examination. Both in health and in disease the bodily temperature presents regular variations in the course of twenty-four hours. Usually there is a decrease of temperature beginning about one or two o'clock in the morning, reaching a *thermic minimum* grade between six and nine in the morning. This is called the *morning remission.* From about one in the afternoon the temperature rises forming the *evening* exacerbation, reaching its minimum grade between three and six P. M. This necessitates the taking of thermometric measurement twice in the twenty-four hours, and should be as near the same hour as possible. A number of observation tables have been arranged by several authors, which readily tabulate all daily observations. Comparison of diurnal fluctuation made several days in succession readily indicates the type of fever to be treated. Extreme temperatures above and below the normal indicate great derangement of vital functions. Very high temperatures, especially if continued in the body for any length of time, cause exceeding alterations in the tissues and enormously reduce their vitality. It is, indeed, a sure forebodeing of speedy death. The same may be said of very low temperatures, yet which may exist, if not excessive, for quite a while in the body and not preclude a possibility of recovery.

The following scale of fever temperatures, according to WUNDERLICH, have been adopted: Normal temperature in adult males: 37.0° to 37.4° centigrade; subfebrile, 37.5° to 38.0° centigrade; febrile temperature, light grade, 38.0° to 38.4° centigrade; moderate grade, 38.5° to 39.0° centigrade in the morning, 39.5° in the evening. Considerable degree of fever: 39.5° morning, 40.0° centigrade in the evening. High grade fever: 39.5° to 39.9° centigrade in the morning, 40.5° to 40.9° centigrade in the evening. When the temperature rises above 41.5° centigrade, the prognosis of the case is considered very unfavorable. With 42.5° centigrade, death is generally sure.

The last-mentioned high temperatures are called *hyperpyretic*, and may terminate favorably when it exists in the body a short time. *Hirz*, of Strassburg, mentions a case of intermittent with a temperature of **44.0**° centigrade, = (**111.1**°) Fahrenheit. *Toele* claims to have observed a case of fever following a fractured lower rib with a temperature of **55.0**° centigrade, or **131.0**° Fahrenheit. In cases with temperatures of 40.0° centigrade increase of every fraction of a degree denotes increased danger. In fever as in health the temperature of the body varies at the same hour of the day. There are cases of fever in which there is inversion of the regular diurnal variation of temperature so that the high grade falls in the low grade period. *Traube* has called this the *typus inversus. Burniche* and *Debeczky* have met with it in most cases of pulmonary consumption. *Eichhorst* found it in two cases of convalescence from typhoid fever. *Griesinger* and *Alvornega* met with fever cases where the excerbations existed at midnight and the days were free from fever. The difference of diurnal fluctuations of temperature determines the type of fever, of which four types have been adopted. These are: the *continued*, the *remittent*, the *intermittent*, and the *recurrent fevers.* In continued fevers the difference in the diurnal variation of temperature does not exceed 1.0° centigrade, the mean height being 39.0° centigrade. In remittent fevers the diurnal oscillation is above 1.0° centigrade; usually fluctuating between one and three degrees centigrade. In hectic fever the variation is often very great, the rise being near forty, the fall sometimes below the normal. The latter usually exist in cases of pyæmia and septecæmia, and in some latter stages of abdominal typhoid. Intermittent and recurrent fevers form a type *sui generis.* The intermittents are produced, according to *Klebs* and *Tomasi* by the *Bacillus Malaria*, the recurrent by *Spirillium Obermeyeri.* As a rule, in intermittent the attacks begin with chills and rigors, when the internal temperature is very high, and are followed by more or less high fevers, of variable duration, usually ending with perspiration. When such attacks occur daily they are said to belong to the *quotidian type.* When there is an interval of forty-eight hours between the attacks they are of *tertian type.* With an interval of seventy-two hours they are of *quartan type.* In recurrent fevers the chill is followed by continued fever of more or less high grade for about five or eight days. An interval of freedom from attacks for about seven or eight days and a recurrence of attack of chills with fever continued for about five or seven days, and ending under critical phenomena. During the fever stage there exist sometimes very high temperatures. The alternate attacks and intervals of freedom from fever may continue for some time.

Although the factors which determine the quantity of animal heat widely differ in the production of its sum total during the several periods of life, yet the constancy of the bodily temperature is maintained by a nicely balancing mechanism, with exception of an exceedingly slight variation, throughout all its period. In childhood the enhanced quantity of produced heat is balanced by increased Elimination of caloric. Adult life regulates its temperature to a nicety, and in old age, with its reduced power of heat-production, there exists parallel to it reduction of eliminatory power. In childhood and in old age, from the naturally lesser powers of resistance to all vicissitudes of life, an excessive deviation from the normal temperature is more dangerous than in robust adult life. Thus a rise of temperature which might prove only a distant danger to an adult is often fatal to an old person. Whilst on the other hand reduced bodily temperature, which might be borne by an adult, and especially by an aged person with impunity, becomes at once fatal to a child, especially to one in the earliest period of life. Like all other functions the heat regulation of the body depends distinctly on the nerve centra, and these require gradual development and steady increase of its functional power, which is itself only brought about by the growth and development of the body from childhood to mature age.

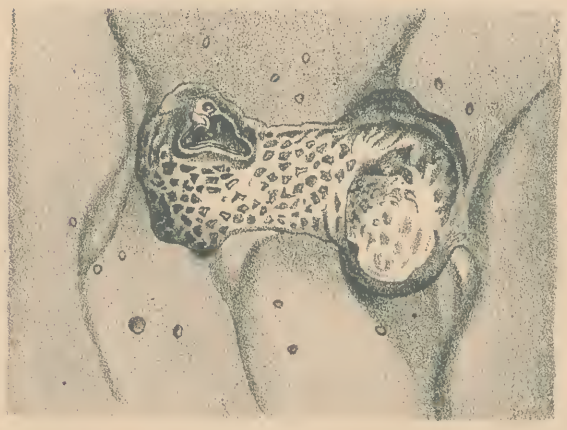
MORBID HISTOLOGY IN ABDOMINAL TYPHOID AND OTHER INFECTIOUS DISEASES.

Sec. X. *Tab. IV.*

Fig. 1. a

Fig. 1 b.

Fig. 2.



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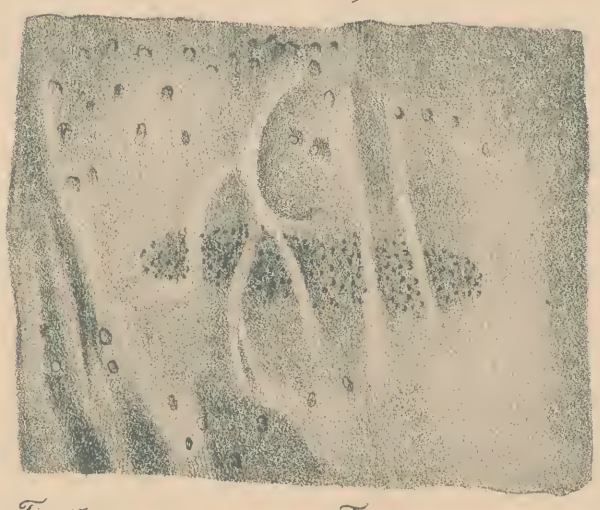

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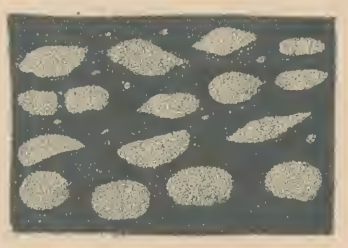

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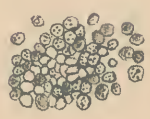

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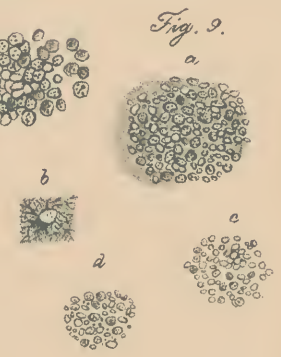

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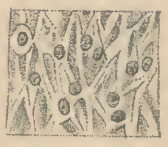

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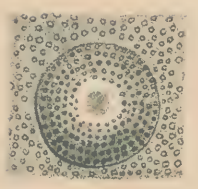

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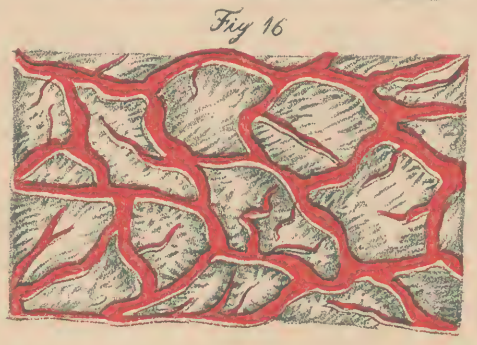

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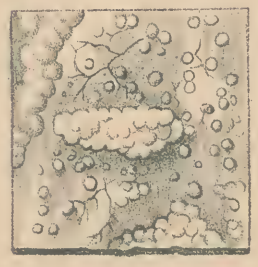

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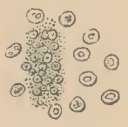

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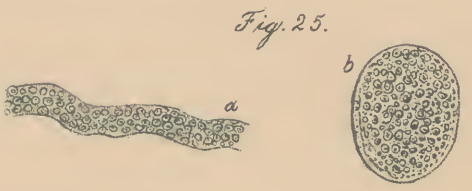

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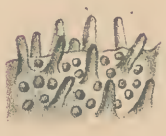

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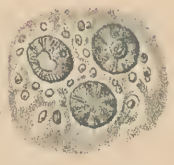

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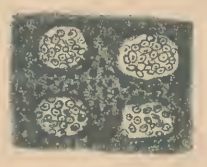

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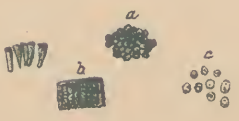

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JEANCON'S PATHOLOGICAL ANATOMY